DISSERTATION ON

"EVALUATION OF PRE-OPERATIVE AND POST OPERATIVE POLYSOMNOGRAPHY FOR OBSTRUCTIVE SLEEP APNEA"

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BONAFIDE CERTIFICATE

This is to certify that this dissertation is a bonafide record of work done by Dr.M.SIVARANJANI on **"EVALUATION OF PRE-OPERATIVE AND POST OPERATIVE POLYSOMNOGRAPHY FOR OBSTRUCTIVE SLEEP APNEA"** during her M.S. ENT course from April 2014 to April 2017 at the Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai. She is appearing for her M.S (ENT) Branch – IV Degree Examination in April 2017 and her work has been done with partial fulfillment of the regulations of The TamilNadu Dr. M.G. R Medical University, Chennai. I forward this to

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DECLARATION

I, **M.SIVARANJANI**, solemnly declare that this dissertation entitled“**EVALUATION OF PRE-OPERATIVE AND POST OPERATIVE POLYSOMNOGRAPHY FOR OBSTRUCTIVE SLEEP APNEA”**is a bonafide work done by me in Upgrade Institute Of Otorhinolaryngology, Madras Medical College and Rajiv Gandhi General Hospital, Chennai during the period of 2014 to 201 under the guidance of **Prof.Dr.M.K.RAJASEKAR M.S.D.L.O.,** Director and Professor, Institute Of Otorhinolaryngology, Madras Medical College and Rajiv Gandhi General Hospital, Chennai and submitted to The Tamilnadu Dr.M.G.R.Medical University, Guindy, Chennai – 32 in the partial fulfillment of the regulations for the award of the M.S.E.N.T ., (Branch IV).

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ABBREVIATIONS

OSA	:	Obstructive Sleep Apnea
OSAHS	:	Obstructive sleep apnea hypopnea syndrome
AHI	:	Apnea Hypopnea Index
TFT	:	Thyroid Function Test
EEG	:	Electroencephalagram
EMG	:	Electromyogram
EOG	:	Electro occulogram
REM	:	Rapid Eye Movement
NREM	:	Non Rapid Eye Movement
SWS	:	Slow Wave Sleep
RERA	:	Respiratory Effort Related Arousal
CPAP	:	Continuous Positive Airway pressure
BMI	:	Body Mass Index
UPPP	:	Uvulopalatopharyngoplasty
DISE	:	Drug Induced Sleep Endoscopy

ESS	:	Epworth Sleep Scale
PSG	:	Polysomnography
TFT	:	Thyroid Function Test
SDB	:	Sleep Disordered Breathing
AASM	:	American Academy of Sleep Medicine
SL	:	Sleep Latency
TST	:	Total Sleep Time
SE	:	Sleep Efficiency
VPI	:	Velopharyngeal Insufficiency
TRT	:	Total Recording Time
RAS	:	Reticular activating system
ESP	:	Expansion Sphincteroplasty
HMS	:	Hyoidmyotomy with suspension
MMP	:	Maxillo-mandibular advancement procedures

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INTRODUCTION

Obstructive sleep apnea, an entity of sleep disordered breathing is characterized by repeated episodes of narrowing or collapse[1] of pharyngeal airway during sleep resulting in substantial reduction/ complete cessation of airflow despite ongoing breathing efforts. It is a chronic and potentially serious disorder affecting 2-4%[2] of individuals with serious complications like hypertension, diabetes, stroke, myocardial infarction and neurocognitive effects[3]. Though the OSA patients are classically treated with lifestyle modifications and medical management, compliance is poor in these patients and thus warranting surgical management. Surgical modification of airway has been performed since decades but with inconsistent results. With current knowledge in understanding the pathophysiology and modern investigations surgery has been carried out with the intention of creating more open airway.

In our study after vigorous investigations in OSA patients, various surgeries has been done addressing the level of obstruction and improvement after surgery both in terms of subjective and objective results were analysed.

AIM AND OBJECTIVES OF THE STUDY

1) To study the pre operative and post operative apnea hypopnea index and oxygen saturation.

2) To study clinical improvement post operatively.

3) To study the effectiveness of various surgeries for obstructive sleep apnea.

HISTORY

Obstructive sleep apnea although gained its importance in recent years, there are strong hints of widespread existence as early as 19th century.

1850 - Periodic breathing in sleep was first reported

1870 - British physicians reported on several cases of obstructed apneas as “fruitless contractions of the inspiratory and expiratory muscles against glottic obstruction with accompanying cyanosis during sleep”

1837 - Day time somnolence associated with obesity was first described by “CHARLES DICKENS” in 1837 through his writing in “The Posthumous Papers of the Pickwick club[4]”(fig 1)

1956 - Drs.A.G.Bicklemann, C.S.Burwell and colleagues described “Pickwickian Syndrome”.

Although pickwickian syndrome was described it is only Gastaut et al. in mid 1960 who described “obstructive sleep apnea in obese subjects as intermittent airway obstruction with frequent arousals, thereby providing the first comprehensive links between obesity, sleep-induced airway obstruction, sleep fragmentation, and daytime sleepiness.”

1965 -First polysomnograph recorded apneas during sleep.

1970 - First sleep clinic was established at Stanford University by William Dement

1970 –Elio Lugaresi described completely about “OSA syndrome”.

1983 – Riley described cephalometric evaluation for OSA.

1985 – Fujita described “Uvulopalatopharyngoplasty (UPPP)”.

1986 – Colin Sullivan, a young Australian medical researcher developed nasal “Continuous Positive Airway Pressure”.

1990-Dr.Murray John developed ESS

Figure 1: Pickwick

SLEEP

Sleep is defined as a “temporary state of unconsciousness that can be interrupted by external stimuli and it is regulated by RAS”.

THEORIES OF SLEEP

There are two theories governing sleep.

1)Passive theory:

“Discharge from RAS during prolonged hours of wakefulness leading to fatigue of RAS thereby inducing sleep.”

2)Active theory:

1) “Serotonin from raphe fibres” inhibits RAS thereby promoting sleep
2) “Melatonin from pineal gland” inhibits reticular activating system thereby promoting sleep

STAGES OF SLEEP

The EEG pattern recorded during sleep varies in a cyclic fashion, which repeats in about every 90 minutes. There are about four cycles in normal 6 to 8 hours of sleep.

In normal individuals, sleep cycle begins with slow-wave sleep or Non-REM sleep. There are four stages of slow wave sleep (stages 1 to 4). A person when falls asleep, passes sequentially through these four stages

of increasingly deep sleep and then into REM period. With completion of REM phase, sleep cycle completes. The REM phase is followed by the next new cycle, i.e. with stage 1 of non-REM sleep. Thus, the cycle repeats in every 70 to 90 minutes. Throughout the night, people wake up briefly (called stage W) but are typically unaware of being awake.

EEG FEATURES OF SLEEP

In 1953, Aserinsky, Dement and Kleitman through EEG and polygraphic analysis described different phases of normal sleep characterized by EEG, autonomic and endocrine changes.

During wakefulness, EEG usually shows desynchronized, high frequency, low amplitude known as beta waves in the range of 14 – 30 Hz. During quiet rest with eyes closed, waves range from 8 to 12 Hz, i.e. alpha waves. When we are asleep, we enter into several different states including theta & delta waves which are much slower than those in the awake state.

NREM SLEEP:

When we fall asleep it enters into NREM sleep which is further subdivided into four stages.(fig 2)

Stage 1(transistional stage):

Characterized by

a) Loss of alpha activity

b) Appearance of a low voltage mixed frequency EEG pattern with prominent theta activity (3-7 cps) and occasional vertex sharp waves may also appear.
c) Eye movements become slow and rolling
d) Relaxation of tone of skeletal muscle occurs
e) Motor activity may persist for a number of seconds.Occasionally sudden muscle contractions can occur.

Stage 2:

After a few minutes of stage 1, sleep usually progresses to stage 2 which is the first stage of real sleep occurring about 20 min.

Chracterised by

a) **K complexes** - A typically large and slow (2Hz of slower) EEG wave starting with a negative sharp wave followed immediately by a positive wave.
b) **Sleep spindles** - Short bursts of 12-14Hz lasting between 0.5 and 1.5 seconds

These phenonmenon occur with a background of mostly theta and some scattered delta wave activity.

Stages 3 and 4:

a) Stage 2 is generally followed by a period comprises of stages 3 and 4. Slow waves (< 2 cps in humans) appear during these

stages, which are subdivided according to the proportion of delta waves in the epoch.

b) In stage 3,there is a minimum of 20% and not more than 50% of the epoch time occupied by slow waves.

c) In stage 4 there is greater than 50% of the epoch showing slow wave activity.

Stage 3 and 4 are also referred to as slow wave sleep (SWS), delta sleep, or deep sleep, since arousal threshold increases incrementally from stages 1 through 4. Eye movements cease during stages 2-4, and EMG activity decreases further.

REM *SLEEP*:

Also called as paradoxical sleep. In infants it is called as active sleep because of phasic muscle twitches

Characterized by

a) “Activated” or “desynchronized” EEG (relatively low voltage mixed frequency).

b) Bursts of rapid eye movements.

c) Suppression of EMG activity in skeletal muscles.

REM sleep can be divided into

a) Tonic(persistent)

b) Phasic (episodic) components.

Tonic aspects of REM sleep include “the activated EEG similar to that of stage 1, which may exhibit increased activity in the theta band (3-7 cps) and a generalized atonia of skeletal muscles except for the extraocular muscles and the diaphragm.”

Phasic features of REM include “irregular bursts of rapid eye movements and muscle twitches.”

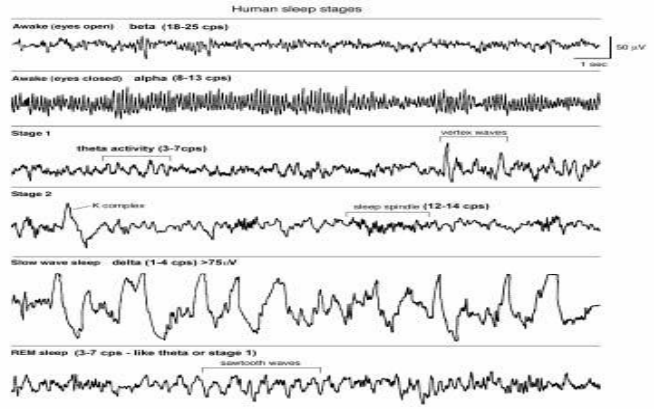


Figure 2: EEG in different stages of sleep

ORGANISATION OF SLEEP

Sleep architecture varies with age. Healthy adult sleeps around 7 to 9 hours per day which decreases progressively as age advances.

Of which

5% in stage 1

50% in stage 2

20-25% in each of SWS (stages 3 and 4) and REM sleep.(fig 3)

Sleep occurs in cycles of NREM-REM sleep, each lasting approximately 90- 110 minutes. SWS (stages 3 and 4) is most prominent early in the night especially during the first NREM period and diminishes as the night progresses. As SWS wanes, periods of REM sleep lengthen, while showing greater phasic activity and generally more intense dreaming later in the night.

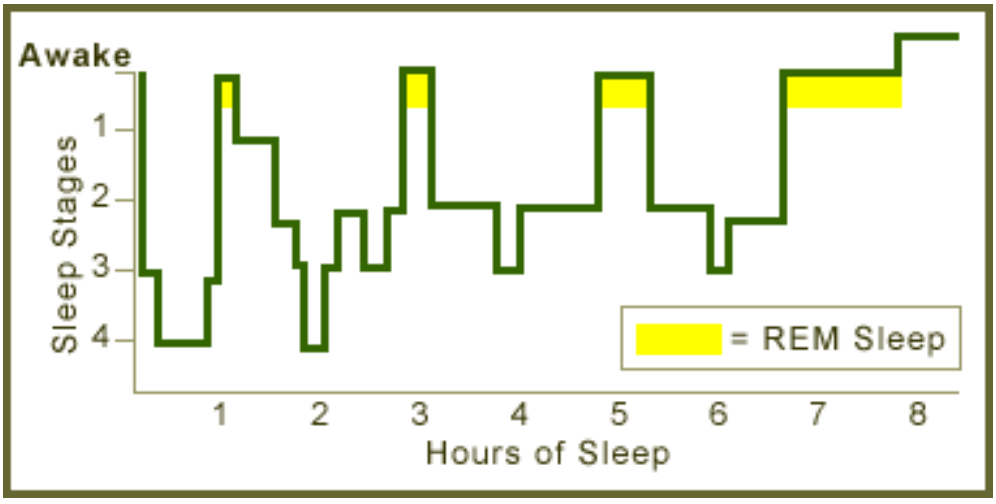


Figure 3: Times spent in different stages of sleep

REGULATION OF SLEEP[5]

Sleep depends on interaction between

1) Intrinsic sleep drive

2) Ultradian REM sleep rhythm

3) Adaptive sleep drive

4) Circadian rhythm

Intrinsic sleep drive

Increases with time spent awake and decreases as soon as sleep occurs

Ultradian rem sleep rhythm:

Has a 90 min cycle of wakefulness and sleep and is independent of intrinsic drive

Adaptive sleep drive:

It occurs as a variety of behavioural response to environment and reflex response to sensory stimulation like light , noise, temperature etc

Circadian rhythm:

Under the control of suprachiasmatic nuclei in hypothalamus which responds in relation to changes in light stimulation.

ANATOMY OF STRUCTURES CONTRIBUTING COLLAPSE

In OSA patients, there is collapse of different pharyngeal soft tissue structures especially that of velopharynx, oropharynx, and /or hypopharynx in addition to soft palate vibrations. Based on the different sites of pharyngeal collapse,

"OSA patients are structurally classified as[6](fig 4):

Type-1 Narrowing or collapse in the retropalatal (velopharyngeal) region alone .

Type-2 Narrowing or collapse in both retropalatal and retroglossal regions .

Type-3 Narrowing or collapse in the retroglossal region alone" .

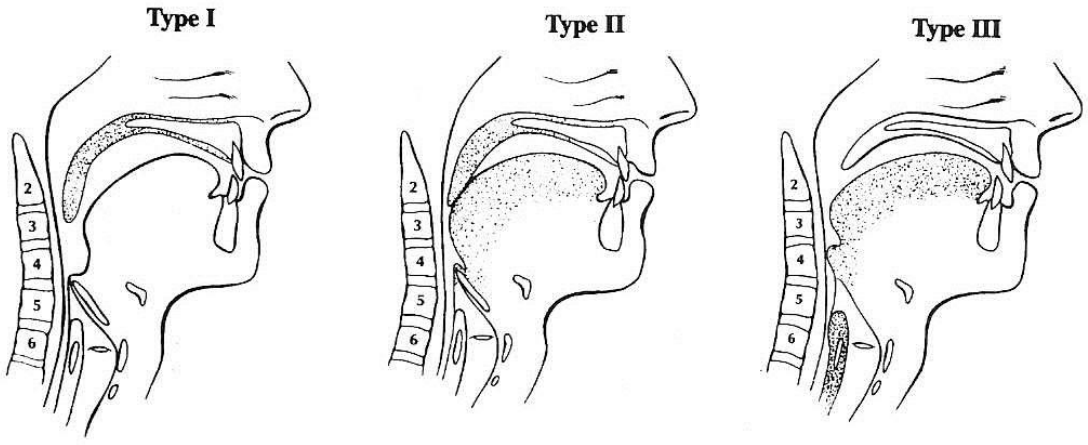


Figure 4: Sites of collapse

VELUM

The velum or soft palate is more complex anatomical structure made up of palatal aponeurosis, fat, lymphoid tissue, and muscles(fig 5)

1) Tensor veli palatine

2) Levator veli palatine

3) Palatoglossus

4) Musculus uvulae

5) Superior fibres of superior constrictor muscle.

Velum plays a important role in deglutition, swallowing, blowing, speech(vowels, non nasal consonants).During deglutition soft palate is made taut which makes the tongue to press against the soft palate thereby allowing food bolus to squeezed into oral cavity.Then soft palate is elevated postero-superiorly to press against the posterior pharyngeal wall by the action of levator veli palate to prevent food entering nasal cavity. In addition closure is augmented by medial convergence of lateral pharyngeal wall.

Action of muscles

1) ***Tensor Veli Palati:*** Tensor of soft palate and opens ET while swallowing and yawning.

2) ***Levator Veli Palati:*** Elevation of soft palate.

3) ***Palatoglossus:*** Elevation of posterior part of tongue and approximates soft palate and tongue.

4) ***Palatopharyngeus:*** Elevates the pharyngeal wall anterosuperiorly and medially during deglutition.

5) ***Musculus Uvulae:*** To pull the uvula up and shorten it.

"All muscles of soft palate are innervated by pharyngeal plexus except tensorveli palate muscle which is innervated by trigeminal nerve."

PALATOPHARYNGEAL SPHINCTER(PASSAVANT'S MUSCLE)

"It arises from anterosuperior surface of palatine aponeurosis to blend with upper border of superior pharyngeal constrictor to encircle the pharynx as a sphincter. It acts along with palatopharyngeus and levator palate to help in closure of pharyngeal isthmus[7,8]".

MUSCLES OF TONGUE

Intrinsic muscles:

1) Superior longitudinal muscles

2) Inferior longitudinal muscles

3) Transversus linguae

4) Verticalis linguae

Extrinsic muscles

1) Styloglossus-Pulls tongue upwards and backwards.

2) Palatoglossus-Muscles of both side acting together bring the palatoglossal arches together closing the aperture from oral cavity to pharynx.

3) Genioglossus-Protrudes tongue (safety muscle of tongue)

4) Hyoglossus-Depresses tongue

"All muscles of tongue are supplied by hypoglossal nerve except palatoglossus which is supplied by cranial part of accessory nerve".

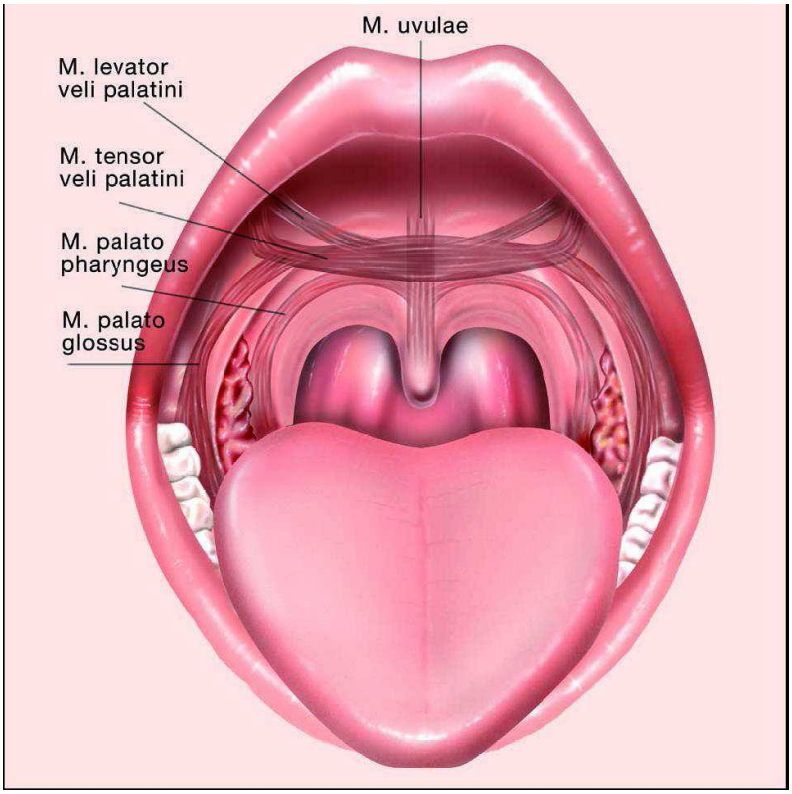


Figure 5: Muscles of soft palate

PATHOPHYSIOLOGY

Obstructive sleep apnea/hypopnea syndrome (OSAHS) is a common disorder affecting 2–4% individuals. It is characterised by “recurrent sleep induced collapse of the pharyngeal airway leading to hypoxaemia and hypercapnia, with arousal from sleep thus re-establishing patency of airway”. Though the pathophysiology of OSAHS is incompletely understood, it is believed that OSAHS patients have an anatomical predisposition to airway collapse and their patency is maintained during wakefulness by mechanisms leading to increase in the activity of pharyngeal dilator muscles. These protective mechanisms fail during sleep, leading to collapse of the pharyngeal airway behind the palate, tongue, or both.

MECHANICS OF THE PHARYNX:

The pharyngeal airway is a complex structure with many functions including respiration, speech, and swallowing. To accomplish all these functions the evolution of the structure and function of the pharynx has required certain‘‘trade offs’’. It has been studied that during evolution of speech in man, rigid support of the hyoid bone to the pharynx which is present in many mammals is lost since speech required substantial laryngeal mobility and thus depending mainly on pharyngeal dilator muscle activity to ensure its patency. However, this pharyngeal dilator muscle activity is highly variable in each individual which is a product of

the intrinsic anatomy and collapsibility of their airway. Thus individuals with an anatomically large airway are less dependent on pharyngeal muscles to maintain airway patency than those individuals with a smaller airway. When the pharyngeal collapse does occur, it usually occurs either in the velopharynx (behind the soft palate), the oropharynx (from the tip of the soft palate to the epiglottis), or both.

Human pharynx can be designed as a collapsible tube, the patency can be described using a ‘‘**balance of pressures’’**[9] concept. The upper airway size depends on the balance between two following forces.(fig 6,7)

“Force that collapses the airway tissue (such as negative intraluminal pressure and increased extraluminal pressure)

Force that maintains the patency of airway(contraction of pharyngeal dilator muscles)”.

The transmural pressure of the pharynx (Ptm) is thus “equal to the pressure in the lumen (Pl) minus the surrounding pressure in the tissue (Pti), with the airway lumen becoming smaller as Ptm decreases”.

The change in area for a given change in pressure gives the effective elastance of the pharynx. The Ptm at which the area of the pharynx equals zero is the closing pressure of the pharynx. Using this model it is measured that normal individuals have the critical closing pressure (Pcrit, pressure at zero flow) below 28 cm H2O during sleep

while those with mild OSAHS/snoring have a slightly negative Pcrit value and those with severe disease have a Pcrit of 0[10]. This suggests that in patients with apnea an anatomically predisposed smaller airway is more prone to collapse during sleep when muscle activity may be low.

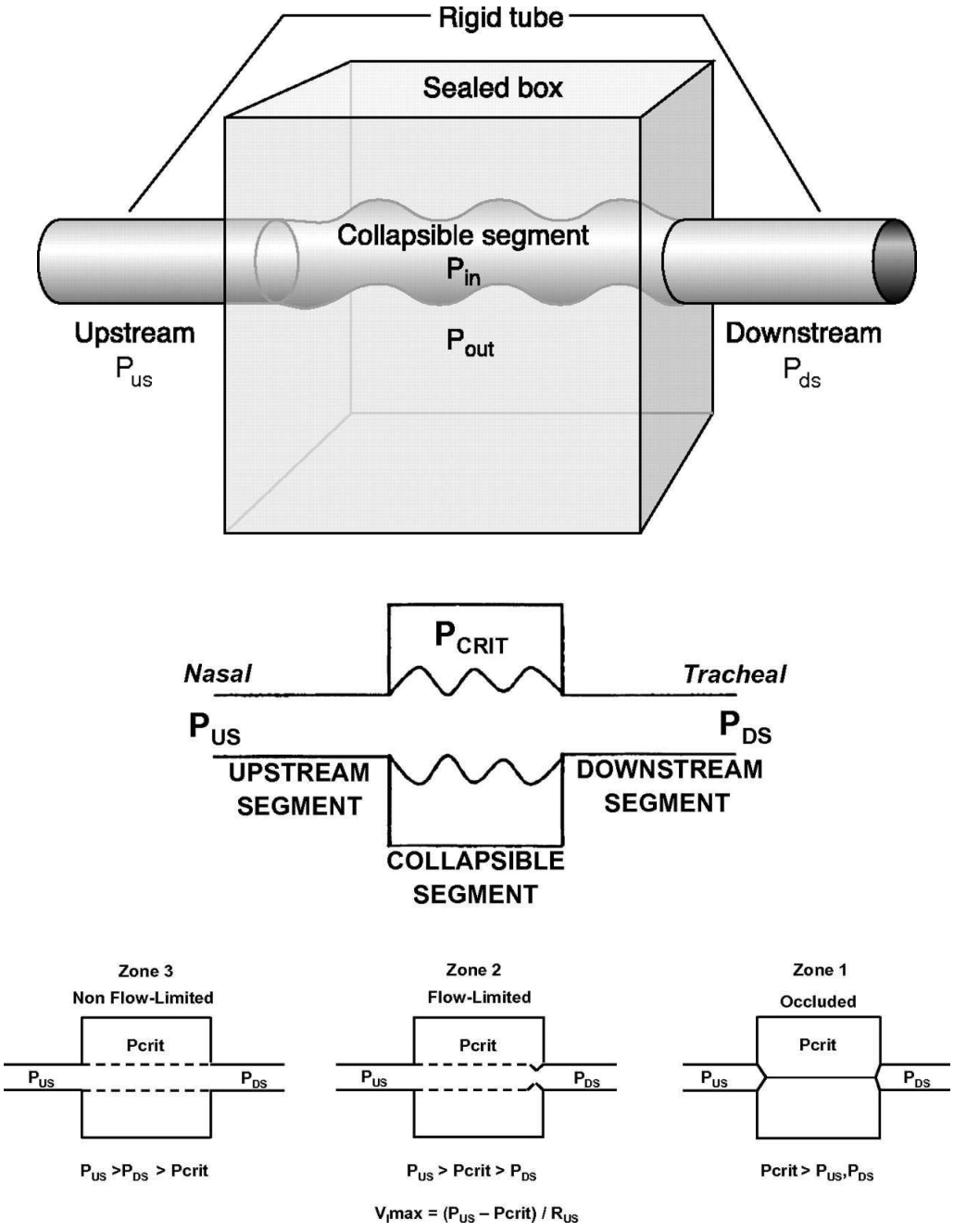


Figure 6,7: Balance of pressure concept

VARIANT ANATOMY IN OSA PATIENTS:

Normal individuals and patients with OSAHS have considerable difference in the anatomy of pharyngeal airway which predisposes them to have increased airway resistance and more chance of airway collapsibility.

OSAHS patients have

1) Reduction in the length of the mandible

2) Inferiorly positioned hyoid bone

3) Retroposition of the maxilla

4) Soft tissue abnormalities like increase in the volume of the tongue, soft palate, parapharyngeal fat pads, and the lateral walls surrounding the pharynx.

5) Long axis of airway is directed anterior-posterior rather than laterally.

6) Increased length between hyoid and mandible

All the above factors are either genetically determined or due to obesity related changes.

PHARYNGEAL MUSCLE FUNCTION AND THE EFFECT OF SLEEP:

The muscles controlling airway patency fall into three groups:

1) Muscles influencing hyoid bone position(geniohyoid, sternohyoid)

2) Muscle of the tongue (genioglossus)

3) Muscles of the palate (tensor palatini, levator palatini).

These muscles can be divided into two groups

a) PHASIC: Muscles which have increased activity during inspiration and so stiffening and dilating the upper airway thus counteratcing the collapsing influence of negative airway pressure.Of which genioglossus is the most important phasic muscle. In expiration when the pressure becomes positive the activity of these muscles is substantially reduced (although not eliminated.[11]

b) TONIC: Muscles have a relatively constant level of activity throughout the respiratory cycle called tonic or postural muscles. Ex: tensor palate. They also play a role in the maintenance of airway patency[12].

The activity of the pharyngeal dilator muscles depends on number of variables

1) Motor nuclei in ventral medulla receive input from central respiratory pattern leading to activation of the genioglossus muscle(inspiratory phasic activation) before the onset of diaphragmatic contraction or inspiratory airflow, thus ‘‘preparing’’ the upper airway for negative pressure.

2) Rising PCO2 and falling PO2 can augment the activity of these muscles through respiratory premotor nuclei

3) Wakefulness drive to these muscles may exist

4) Negative intrapharyngeal pressure is the most important local stimulus to activation of the pharyngeal muscles during wakefulness.There is linear relationship between negative pressure and genioglossus activity(GG EMG and Pepi) which is more pronounced in apneic individuals.

Action of pharyngeal dilator muscle is thus contolled by all the above factors in AWAKE STATE to maintain ventilation. Of which in patients with apnea negative pressure reflex is exaggerated in order to compensate for anatomically deficient airway (NEUROMUSCULAR COMPENSATION)

During transition from AWAKE TO SLEEP

1) Phasic neurones largely maintain their activity during sleep, while tonic neurones have large decrements in activity and thus the inspiratory phasic activity of muscles falls initially and then recovers to waking state but tonic activity of muscle (the tensor palatini) lose activity at sleep onset which continues to fall as sleep deepens, reaching levels of 20–30% of waking values[13].

2) As of all reflexes which gets reduced in sleep negative pressure reflex also get decreased in sleep.

Above factors puts the airway in compromised state during sleep. In OSAHS(anatomically compromised airway) patients in addition to above factors, there is loss of neuromuscular compensation leading to greater incidence of airway collapsibility thus leading to apneic episodes.

OTHER FACTORS

LUNG VOLUME

Upper airway size increases at higher lung volumes, because increased lung volume leads to increased ‘‘tracheal tug’’ and thus increasing the size of upper airway and so decreasing resistance to airflow. OSAHS patients have a “greater change in upper airway dimensions with changes in lung volume, that is, GREATER LUNG VOLUME DEPENDENCE OF UPPER AIRWAY SIZE”. In NREM sleep as the lung volume and end expiratory volume falls patients with OSAHS tends to have airway collapsibility as the airway is already compromised.

VENTILATORY STABILITY

Ventilator stability is measured by loop gain. Loop gain is “the measurement of the tendency of the ventilatory control system to amplify respiration in response to a stimulus or perturbation.”

Ventilation will become unstable if the loop gain is >1 will stabilise if the loop gain is <1.

Studies shows that patient with apnea have high loop gain[14] and thus highly unstable respiratory control system.

PROGRESSION OF SLEEP APNEA/HYPOPNEA

Sleep apnea which is initially mild in individuals worsens over time because of the following reasons:

1) Snoring and repeated upper airway occlusion

↓

Edema and swelling of upper airway soft tissue structures

↓

Further narrowing of the upper airway

↓

Airway collapse occurs

↓

2) Repeated contraction of dilator muscle, vibratory trauma[15]

↓

Eccentric contraction of muscle and muscle dysfunction

↓

Defect in sensing sensory stimuli leading to collapse

APNEA TERMINATION

Hypoxia, hypercapnia associated with airway obstruction

↓

Increased respiratory effort/ airway pressure becoming negative/ or can directly stimulate RAS

↓

Arousal centre stimulated

↓

Upper airway muscle activity increases followed by relief of obstruction associated with loud snort and hyperventilation.

Then the patient goes in for sleep and the cycle continues. In REM sleep arousal threshold is higher and so the duration of apnea will be longer in REM sleep.

CLASSIFICATION OF OBSTRUCTIVE SLEEP RELATED BREATHING DISORDERS

Sleep disordered breathing is a wide spectrum ranging from simple snoring to obesity hypoventilation syndrome[16]

SNORING

Defined as “sound generated by the vibration of pharyngeal soft tissues”. Usually it is more during inspiration than expiration and it may also present as a separate symptom without any association to day time sleepiness.

UPPER AIRWAY RESISTANCE SYNDROME

UARS is a recent entity which describes “patients with symptoms of OSA and PSG evidence of sleep fragmentation with AHI<5 without oxyhemoglobin saturation”

OBSTRUCTIVE SLEEP APNEA SYNDROME

OSA is defined as “five or more respiratory events (apneas,hypopneas, or RERAs) in association with excessive daytime somnolence,waking with gasping, choking, or breath-holding, or witnessed reports of apneas, loud snoring or both”.

OBESITY HYPOVENTILATION SYNDROME

It is defined as “combination of obesity (BMI>30Kg/m2) ,hypoxemia during sleep, hypercapnia during daytime resulting from hypoventilation”

CLINICAL FEATURES

SYMPTOMS

Patients of OSA presents with nocturnal and daytime symptoms.

Nocturnal symptoms includes “snoring, witnessed apnea, dyspnea, drooling, dry mouth, bruxism, restless sleep/frequent arousal, nocturia”.

Daytime symptoms[17] includes “Excessive day time sleepiness, morning headaches, neuro cognitive impairment, diminished quality of life, mood and personality changes, sexual dysfunction”

SIGNS

“Enlarged and elongated uvula, hyperplastic or thick soft palate, constricted oropharynx, macroglossia, enlarged tongue base, prominent oropharyngeal folds, skeletal deformities(maxillary& mandibular retrognathism or hypoplasia, receded chin), adenoids, obstructive tonsils, deviated septum, enlarged nasal turbinates, nasal polyps or any other obstructive masses”

EVALUATION OF OSA PATIENTS

MODIFIED MALLAMPATTI SCORING

In 1985, mallampatti et al. proposed a grading system to predict difficult intubation by evaluating the relationship between the tongue and other oropharyngeal structures. Mallampatti scoring is assessed with the maximally protruding of tongue.

Friedman et al. modified the mallampatti scoring by assessing without protrusion of tongue as it is the most natural position during sleep to asses the relation of oropharynx in relation to tongue and palate .The MMP has a highly predictive value for assessing the severity of OSA and its outcome after surgical procedures.

"Modified Mallampati classification by Friedman et al[18](Fig 8)

1) **Grade 1** : Tonsils, pillars, and soft palate clearly visible.

2) **Grade 2** : uvula, pillars, and upper pole visible.

3) **Grade 3** : only part of the soft palate visible; tonsils, pillars and the base of the uvula cannot be seen .

4) **Grade 4** : only the hard palate is visible"

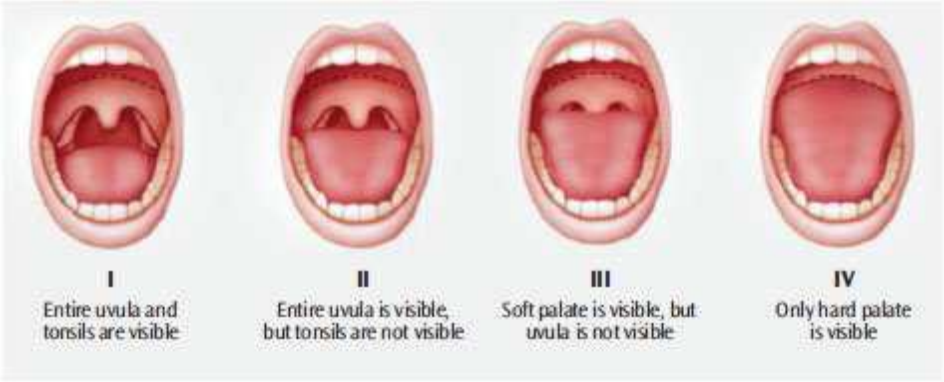


Figure 8: Friedman tongue position

EPSWORTH SLEEPINESS SCALE

Epworth Sleepiness Scale was developed in 1991 by Dr. John W. Murray.He intended to "quantify the likelihood of someone to fall asleep in 8 different common situations". The scores range from 0 to 24 and 10 is the normal limit. The higher the ESS score, the more severe the condition.

1) Sitting and Reading

2) Watching TV

3) Sitting, inactive in a public place (e.g., waiting room, a theater or a meeting)

4) As a passenger in a car for an hour without a break

5) Lying down to rest in the afternoon when circumstances permit

6) Sitting and talking to someone

7) Sitting quietly after lunch without alcohol

8) In a car, while stopped for a few minutes in traffic

0- would never doze off

1- slight chance of dozing off

2- moderate chance of dozing off

3- high chance of dozing off

MULLER MANEUVER (PALATE, BASE OF TONGUE, AND LATERAL WALLS)

The muller's maneuver was developed to evaluate various sites and severity of collapse of upper airway . Procedure involves "viewing the upper airway using the nasopharyngoscope at rest and with the maximal inspiratory effect against a closed nose and mouth. Soft tissue collapse especially the base of tongue, lateral pharyngeal walls and palate are examined.This collapsibility is rated from 0(minimal collapse) to 4+ (complete collapse)" .Muller maneuver score is important in preoperative evaluation of SDB severity. However, the repeatability of this maneuver is very low. Palatal and lateral wall collapse has a greater correlation, but with base of tongue it is low.

POLYSOMNOGRAPHY

It is the gold standard investigation in assessing the sleep disordered breathing and assessing the effectiveness after surgery.

PARAMETERS

PSG monitors many physiological signals that is required for the assessment of sleep disordered breathing. These signals are displayed in graph in organized waveform.

a) Sleep staging

b) Respiratory airflow

c) Respiratory effort

d) Pulse oximetry

e) Ventilation

f) Cardiac rhythm

Sleep staging

ECG, EMG, EEG are the parameters monitored in sleep staging.

Sleep staging helps in

1) Assesing the severity of the disease(it adjusts the total no of respiratory events based on total sleep time)

2) Helps in identification of respiratory events associated with arousal

Respiratory Airflow

It is monitored by two methods

a) Oronasal thermistory

b) Nasal pressure transduction.

a)Oronasal thermistory

Helps in identification and scoring of apneas.It is the superior modality in detecting low levels of airflow.

MECHANISM: Exhaled air is warmer than the environmental air since the air is heated in the lungs. Oronasal thermistory uses this mechanism and “measures fluctuations in air temperature at nose and mouth” and thus measures even modest level of airflow.

b)Nasal pressure transduction

It is superior in measuring “subtle changes in airflow as seen in hypopneas”

MECHANISM:Negative pressure fluctuation determines inspiration and positive pressure fluctuation determines expiration.Nasal pressure transduction uses this mechanism and detects airflow by measuring variations in air pressure.

Respiratory effort

Measuring respiratory effort helps in distinguishing central and obstructive apnea. It can be measured in following ways(RIP, Piezoelectric belts, PVDF, Esophageal manometry)

Pulse oximetry

It measures arterial oxyhaemoglobin saturation.

MECHANISM:Uses “DUAL WAVELENGTH”(red and infrared). Light transmitter and receiver is placed around the arterial bed and the “ratio of red and infrared transmission” is used to calculate spo2.

Ventilation

It is required only in children. In adults, only if hypoventilation is suspected ventilation is monitored.

METHODS

1) Transcutaneous co2 measurement

2) End tidal co2 monitoring

Cardiac rhythm

Monitored by ECG. Single lead is sufficient to detect arrhythmias. Usually lead 2 is used.

TYPES OF PSG

Depending upon the parameters measured PSG are of four types[19].

Level I - “Standard PSG with minimum of seven parameters measured (EEG, EOG, chin EMG,ECG, airflow,respiratory effort, and oxygen saturation) in a specilist unit”.

Level II - “Comprehensive portable PSG same, except that a heart rate monitor can replace the ECG and a technician is not in constant attendance”

Level III - “Modified portable sleep apnea testing is a cardiorespiratory study , minimum of four parameters ventilation (at least two channels of respiratory movement, or respiratory movement and airflow), heart rate or ECG, and oxygen saturation”. Done at home.

Level IV - “Continuous (single or dual) bioparameter recordings where devices that measure a minimum of one parameter, usually oxygen saturation are utilized”

IDENTIFICATION AND SCORING OF EVENTS

GENERAL PRINCIPLE

1) Identification of apneic events on PSG requires measurement of airflow and confirmation by EEG that the event occurred during sleep.

2) Respiratory effort measured to differentiate central and obstructive type.

3) Oximetry measurement improves the reliability and also helps in scoring type of events.

Arousal:

Identified by

“Abrupt shift of EEG frequency including alpha, theta frequency of >1 hz that lasts atleast 3 sec with atleast 10 sec of preceding stable sleep”.

In REM sleep there should also be increase in the activity of submental EEG lasting atleast 1 sec.

AROUSAL INDEX: Total no of arousal divided by total sleep time.

Apnea

Apnea is defined as cessation/ near cessation of airflow.

It is detected by recording event of "90% or greater reduction in airflow for a period of 10 sec".

It is classified as

a) OBSTRUCTIVE: associated with respiratory effort throughout the event

b) CENTRAL: absence of respiratory effort

c) MIXED: absence of respiratory effort in initial part later followed by appearance of respiratory effort.

Hypopnea

Hypopnea is defined as " reduction of airflow to a degree that is insufficient to meet the criteria of apnea".

According to AASM, hypopnea is

a) "Airflow decreased by atleast 30% compared with previous baseline.

b) Diminished airflow for atleast 10 sec.

c) Event is associated with either 3 oxygen desaturation/ EEG arousal."

It can either be central/ obstructive. When hypopnea is associated with features of upper airway narrowing like thoracoabdominal paradox, snoring etc it is obstructive type. Otherwise it is central hypopnea.

Respiratory effort related arousal(RERA)

Change in airflow that doesn't meet criteria of apnea and hypopnea is called as RERA.

It is defined as "event lasting for atleast 10 sec and associated with flattening of nasal pressure waveform and/or there is evidence of increasing respiratory effort terminating in an arousal but not meeting criteria for apnea or hypopnea".

INTERPRETATION FROM POLYSOMNOGRAPHY:

From the above parameters severity of the sleep disordered breathing can be derived by many indices.

a) Apnea hypopnea index

b) Respiratory disturbance index

c) Oxygen desaturation index

Apnea-Hypopnea index(AHI)

It is calculated as “number of apneas and hypopneas per hour of total sleep time”.

“NORMAL : AHI<5

MILD OSA : AHI 5-15

MODERATE OSA : AHI 16-30

SEVERE OSA : AHI>30”[20]

It can further be subdivided into obstructive AHI and central AHI.

Respiratory disturbance index(RDI)

It is derived by “adding total no. of apnea, hypopnea, RERA and divided by total sleep time in hours”.

Oxygen desaturation index(ODI)

It is defined as “total no of times per hour of sleep that a patient’s oxyhaemoglobin saturation drops by 4 percent from baseline”.

SLEEP HYPNOGRAM

A sleep hypnogram is a summary of the entire night’s PSG data in a graphic form. It gives a good snapshot of sleep architecture, distribution of respiratory events, and oxygen saturation trends in different sleep stages,sleep position, and at different times of the study night. It is helpful to open a window for the hypnogram simultaneously while reviewing the PSG.

The most important parameters measured in sleep hypnogram are:

1) Total recording time(TRT)-“ beginning and end of recording”

2) Total sleep time(TST)-“ actual sleep REM +NREM”

3) Sleep period time(SPT)- “ sleep onset to final awakwning”

4) Sleep efficiency (SE)-“ percentage of TST in total time in bed”. SE>85%is normal

5) Sleep latency (SL) –“time elapsed between lights out to the first epoch of sleep (usually stage 1)”.

FLUOROSCOPY

Fluoroscopy is a readily available technique to assess dynamic airway anatomy and sites of obstruction in OSA patients. Somnofluoroscopy combines fluoroscopy with polysomnography and radiologically evaluate the sites of obstruction during episodes of apnea and hypopnea. Advantages of fluoroscopy include direct observation of obstructive sites during episodes of apnea, and availability of fluoroscopy in most hospitals. Drawbacks include high radiation dose, superimposition of structures, and the possible need for sedation to attain sleep during the procedure.

X RAY CEPHALOMETRY

Lateral x ray cephalometry is a valuable tool in assessing skeletal craniofacial morphology.

DYNAMIC MAGNETIC RESONANCE IMAGING

MRI provides anatomic definition of soft tissue structures, allows for multiplanar imaging, and does not expose patients to radiation. With newer technologies, the dynamic airway can be evaluated with rapid image acquisition; multiple images per second. The main disadvantage of dynamic MRI arises in regards to patient comfort, concurrent sleep evaluation, scanner noise with possible requirement of sedation, and examination expense.

DISE

Drug induced sleep endoscopy is a procedure which involves endoscopic evaluation of upper airway and identifying the level of obstruction in a pharmacologically sedated patients. Results are interpreted as VOTE classification[21].

SITE	AP	LATERAL	CONCENTRIC
Velum			
Oropharynx			
Tongue			
Epiglottis			

DEGREE OF OBSTRUCTION

0 – no obstruction(<50%)

1 – partial obstruction(50-75%)

2 – complete obstruction(>75%)

X – not visualised

TREATMENT

Treatment options for OSA fall under three categories :

1) Life Style Modification

2) Devices that can be worn

3) Surgery

LIFE STYLE MODIFICATION

Weight reduction

Avoiding alcohol and smoking

Sleep posture therapy: lateral or prone position

DEVICES THAT CAN BE WORN

a)Positive airway pressure

Most effective treatment in moderate to severe grades of OSA in tolerant patients which can reverse the negative cardiac and neurocognitive consequences of untreated disease It is available in many forms such as including continuous positive airway pressure (CPAP), adjustable positive airway pressure (APAP), and bi-level positive airway pressure (BI-PAP).

Each delivers positive pressure through a device worn on the face, and serves as an internal pneumatic splint for the airway. CPAP, the most

commonly used form of PAP, typically uses between 5 and 15 cm of water pressure to maintain airway patency. Although CPAP has high efficacy, compliance is limited[22] causing non-adherent to therapy (defined as > 4 hours of use per night)

b)Oral appliances

Mandibular Advancing Device: Advance the mandible anteriorly, which brings forward the tongue and other muscles of the oropharynx and hypopharynx.The position of the palate is also changed with the mandibular repositioning device through action of the palatoglossus muscle.

Tongue Retaining Device (TRD): " It increases pharyngeal patency by pulling the superior aspect of the tongue forward, away from the posterior wall of the pharynx"

SURGICAL MANAGEMENT

Patients with OSAHS who failed to show response to conservative management are treated effectively by surgery with intention to alleviate obstruction and its consequences by creating more open airway. Since the patients with OSAHS may have obstruction at either retropalatal/ retrolingual/ both various surgeries have evolved to address the specific sites of obstruction. Inorder to avoid unnecessary procedures and minimize the surgical interventions a protocol was formulated popularly known Powell–Riley protocol.

It is a two staged procedure addressing specific sites of obstruction(nasal cavity/nasopharynx, oropharynx and hypopharynx).

PHASE 1: Targets soft tissues of upper airway i.e., nose, palate, tongue

PHASE 2: Targets hypopharyngeal and pharyngeal airway by altering skeletal framework.

Powell–Riley protocol surgical procedures[23]:

Phase I

- Nasal surgery (septoplasty, turbinate reduction, nasal valve grafting)
- Tonsillectomy
- Uvulopalatopharyngoplasty (UPPP) or uvulopalatal fl ap (UPF)
- Mandibular osteotomy with genioglossus advancement
- Hyoid myotomy and suspension
- Temperature-controlled radiofrequency (TCRF)-turbinates, palate, tongue base

Phase II

- Maxillomandibular advancement osteotomy (MMO)

- Temperature-controlled radiofrequency (TCRF)-tongue base
- TCRF is usually employed as a adjunct treatment.

INDICATIONS FOR SURGERY

- "Apnea/Hypopnea Index (AHI) >20 events/per hour of sleep
- Oxygen desaturation nadir <90%
- Esophageal pressure (PES) more negative than –10 cm H 2 O
- Cardiovascular derangements (arrhythmia, hypertension)
- Neurobehavioral symptoms (excessive daytime sleepiness [EDS])
- Failure of medical management
- Anatomical sites of obstruction (nose, palate, tongue base)"

CONTRAINDICATIONS FOR AIRWAY SURGERY

- "Severe pulmonary disease
- Unstable cardiovascular disease
- Morbid obesity (BMI >40)
- Alcohol or drug abuse
- Psychiatric instability"

Patients can be categorized into either responders or non responders after surgery.

DEFINITION OF SURGICAL RESPONDERS

1) "Decrease in RDI greater than or equal to 50% and a total of less than 20 events/h

2) Decrease in AHI greater than or equal to 50% and a total of less than 10 events/h

3) Oxygen desaturation nadir 90%

4) Excessive daytime fatigue (EDS) alleviated

5) Normalization of sleep architecture

6) Response equivalent to CPAP on full-night titration"

DEFINITION OF NON RESPONDERS

No change or detoriation in outcome are referred as non responders.

NASAL SURGERIES

Nasal obstruction causes increase in resistance causing increase in negative inspiratory pressure thereby causing airway collapse. Moreover nasal obstruction increases negative pressure and causing functional narrowing of the pharyngeal airway, leading onto hypoxia and sleep apnoea. But nasal surgeries rarely cures OSAHS. In patients undergoing nasal surgeries compliance to nasal CPAP is increased[24] since it reduces CPAP pressures from a mean of 11.9 down to 9.2 centimeters of water pressure by improving nasal breathing.

Septoplasty, septorhinoplasty, and turbinate reduction are the various procedures to correct the underlying nasal pathology.

ADENOTONSILLECTOMY

Adenotonsillectomy is the surgery commonly done in pediatric population to correct loud snoring and restless sleep. The tonsils and adenoids can be removed or reduced in a number of ways like standard cautery, snare, bipolar cautery, harmonic scalpel, coblation, temperature-controlled radiofrequency, or microdebrider-powered shavers. Complications include hemorrhage, pain.

VELOPHARYNGEAL SURGERY

Since 80% of the patients with OSAHS have obstruction at the level of velum many surgeries have evolved to modify the anatomy of velum to maintain airway patency. It can either be minimally invasive or invasive procedures depending upon the severity of the disease. It includes

1) Uvulectomy and laser assisted uvuloplasty

2) CAPSO (Cautery-assisted palatal stiffening operation)

3) Injection Snoreplasty

4) Palatal implants

5) UPPP(uvulopalatopharyngoplasty): UPPP is the most common procedure for the treatment of OSAHS described by Fujita[25] which is a modification of Ikematsu procedure. It consists of

"tonsillectomy, reorientation of anterior and posterior tonsillar pillars, and excision of the uvula and posterior rim of the soft palate"

STEP 1: The incision is marked on the anterior pillars and ventral surface of the palate with electrical cautery

STEP 2: Dissection begins at the right inferior tonsillar pole and anterior pillar and tonsil removed. Incision continued to softpalate leaving dorsal flap. Uvula resected leaving dorsal flap. Incision extended to opposite side in a similar fashion.

STEP 3: Incisions are made at the junction between the dorsal palatal and posterior pillar flaps.

STEP 4: The upper part of the posterior pillar flap is approximated to the ventral palatal mucosa. After removing redundant mucosa soft palate and uvula are sutured.

Complications includes transient/ permanent VPI, pharyngeal symptoms (tightness, dryness, FB sensation in the throat), bleeding, wound dehiscence, prolonged pain, taste and voice disturbance, nasopharyngeal stenosis, detoriation of OSA. Since the complications encounterd are due to the removal of large amount of tissues, new procedures have evolved that reorganizes and preserves tissues and promoting increase in airway. These includes relocation pharyngoplasty,

lateral pharyngoplasty, expansion sphincter pharyngoplasty, Z-palatoplasty, and palatal advancement.

6) Transpalatal Advancement Pharyngoplasty(TAP)

7) Lateral Pharyngoplasty

8) Z-Palatoplasty (ZPP)[26]: Described by Friedman with the aim of removing anterior mucosa only and the splitting of the soft palate in the midline. The key features are the cutting of the palatoglossus muscle, and the sewing of the posterior palatal mucosa to the anterior resection margin, which retracts the midline

STEPS: Outline of the palatal flaps, marked before incision.

↓

The mucosa over the palatal flap is removed and the palatal musculature is exposed

↓

The uvula and palate are split in the midline with a cold knife

↓

The uvular flaps along with the soft palate are reflected back and laterally, over the soft palate.

↓

Two-layered closure of the palatal flaps is done.

Complications include bleeding, temporary and permanent VPI, throat discomfort symptoms like globus sensation, mild dysphagia, dry throat, and inability to clear the throat.

9) Expansion Sphincter Pharyngoplasty: Described by Pang & Woodson[27] in which a "horizontal incision is made in the palatopharyngeus muscle (after tonsillectomy), superolateral incisions are made on the soft palate, in the inferior aspect of the palatopharyngeus muscle is then suspended superolaterally to the arching fibres of palatoglossus, partial uvulectomy performed and then closure of wound is done".Complications are as with other palatal procedures but the incidence is much low.

HYPOPHARYNGEAL SURGERY

Apart from velum, many of the OSA patients have obstruction at the level of tongue base. To address retroglossal area, surgery is directed toward either a reduction in the volume of tongue mass or advancement of the tongue's anterior attachments. Most of the procedures are done as a part of multilevel surgery and not as a solo procedure.

Tongue-Base Reduction Procedures

Tongue-base reduction procedures are intended to reduce the size of the tongue, so that the space between the posterior pharyngeal wall and the tongue base is increased. Procedures include radiofrequency ablation, partial midline glossectomy, lingualplasty, and lingual tonsillectomy,

submucosal minimally invasive lingual excision (SMILE) and transoral robotic glossectomy (TORS).

Radiofrequency tongue ablation:

Radiofrequency tissue ablation which is a common procedure involves the application of a temperature-controlled radiofrequency probe to multiple locations in the base of the tongue. The aim of this procedure is to reduce the tongue volume through scar tissue generation.

Tongue-Repositioning Procedures:

Tongue-repositioning procedures increases the space between the posterior pharyngeal wall and the tongue base by moving the tongue anteriorly by releasing its attachment. It includes tongue suspension, hyoid myotomy and suspension (HMS), and genioglossal advancement.

a)Tongue base suspension: It is designed to stabilize the tongue by anchoring the tongue to the mandible with a permanent suture thereby preventing retrolingual collapse.

b)Hyoid myotomy and suspension (HMS): HMS uses permanent sutures to suspend the hyoid bone to the thyroid cartilage or mandible thereby pulling the tongue anteriorly.

c)Genioglossal advancement: Genioglossus advancement involves mobilization and advancement of the genial tubercle of the mandible, with

limited osteotomy and fixation or forward movement of the lower anterior mandible and attached muscles thereby displacing the tongue anteriorly.

3)Hypoglossal nerve stimulation:

It is a new entity which is neither a tongue base reduction nor a tongue advancement procedure. But it increases retrolingual area by placing an implantable stimulator that stimulates the hypoglossal nerve during inhalation to keep the retroglossal airway open during sleep. Components includes implantable pulse generator, respiratory pressure sensor and stimulation lead.

MAXILLARY MANDIBULAR ADVANCEMENT

Maxillomandibular advancement (MMA) is intended to widen the retroglossal and retropalatal airways by creating LeForte 1 osteotomy to the maxilla and a bilateral split sagittal osteotomy to the mandible to advance both the maxilla and mandible. Though it is recommended as a phase 2 surgery it can be done as a primary procedure in patients with obvious craniofacial issues and multiple sites of upper airway obstruction provided these patients fulfill clinicoradiologic criteria based on cephalometric measurements.

TRACHEOSTOMY

Permanent tracheotomy was the first treatment[28] formulated for OSA and it is still the gold standard surgery for OSA. Because of the morbidity associated with the surgery it is reserved for patients who are

intolerant of mechanical measures or fail upper airway surgery and continue to have severe symptoms or physiologic changes related to obstructive sleep apnea

MULTILEVEL SURGERY

Because of the inconsistent results with various surgeries, friedman proposed a staging system[18].

Stage-I	Friedman tongue position 1 and 2. Tonsil size 3 and 4. BMI <40.
Stage-II	Friedman tongue position 1 and 2. Tonsil size 0, 1 and 2. BMI <40.
	Friedman tongue position 3, 4 tonsil size 3 and 4. BMI <40.
Stage-III	Friedman tongue position 3 and 4. Tonsil size 0, 1 and 2. BMI <40.

OSA patients of stage 2 and 3 have been identified to have obstruction not only at the single level but simultaneously at multiple levels[29] requiring surgery addressing various levels of airway. Therefore multilevel pharyngeal surgery is usually required to surgically overcome the several sites of obstruction.

SURGERY ON EPIGLOTTIS

Rarely patients with OSA have obstruction at the level of epiglottis making it to collapse posteriorly during sleep. To correct this retrodisplacement, epiglottopexy (suspending the epiglottis to the tongue base with a suture) and epiglottoplasty (removing a portion of the superior

part of the epiglottis) are the procedures employed. These procedures are rarely performed, but if performed, they are often done so as part of a multilevel surgery[30].

SURGICAL SUCCESS

DEFINED AS **"as a decrease in respiratory disturbance index greater than or equal to 50% and a total of less than 20 events/h or a decrease in apnea index greater than or equal to 50% and a total of less than 10 events/h.[31]"**

MATERIALS AND METHODS

STUDY PLACE

Rajiv Gandhi Government General Hospital, Chennai – 600003.

COLLABORATING DEPARTMENT

Upgraded Institute of Otorhinolaryngology

STUDY DESIGN

Retrospective and Prospective study

STUDY PERIOD

July 2014 To September 2016

STUDY POPULATION

All patients with snoring and OSA who reported to the upgraded institute of otorhinolaryngology of Madras Medical College during the study period with the fulfillment of inclusion criteria.

INCLUSION CRITERIA

1) Age > 20 years

2) Both sexes (male and female)

3) BMI <40

4) Neck circumference >17 inches for men and >16 inches for women

5) Unsuccessful or refused CPAP therapy

EXCLUSION CRITERIA

1) Age below 20yrs and above 55yrs

2) Hypothroidism and other metabolic disorders

3) BMI >40

4) Associated craniofacial abnormalities

INVESTIGATION

1) Thyroid Function Test (TFT)

2) CT Paranasal sinus

3) DISE (Drug induced sleep endoscopy)

4) Dynamic MRI

ETHICAL COMMITTEE APPROVAL

Institutional Ethical Committee, Government General Hospital, Madras Medical College, Chennai reviewed the experimental design and protocol as well as the letter of information and consent form. Full approval of the board was granted. All patients were given information outlining the experimental protocol and all patients signed a consent form prior to entering the study.

METHODOLOGY

This is a retrospective and prospective study conducted in Upgraded Institute of Otorhinolaryngology, Madras Medical College from June 2014

to September 2016. All patients who attend our op with the complaints of snoring, frequent awakening at night, excessive day time sleepiness, choking in sleep are further evaluated. All the patients underwent clinical examination followed by blood investigations especially thyroid function test and BMI evaluation .Then polysomnography is done with EEG, EOG, ECG, pulse oximetry (Fig-9), oronasal thermistor, thoracoabdominal belts is done to distinguish obstructive or central apnea(fig 10).

Figure 9:Recording of oxygen saturation in PSG

Figure 10:Polysomnographic recordings

Patients with obstructive apnea are further investigated with dynamic MRI, cephalometry, DISE to find the level of obstruction.These patients are given a choice of using CPAP as a treatment for OSA. So patients who defer CPAP and those failed with CPAP are prepared for surgery.

SURGERY

Those patients selected for surgery were admitted in the ward and started with preop antibiotics. Patients shifted to operation theatre. Under general anaesthesia, patient made to lie in supine position with neck extended and mouth wide opened with the help of boyle davis mouth gag. Depending upon the site of obstruction patients underwent surgery accordingly.

ZETAPLASTY

Indication : Circumferential collapse at velum.

After tonsillectomy, incision is made in the palate with the help of cautery(fig 11)**.**

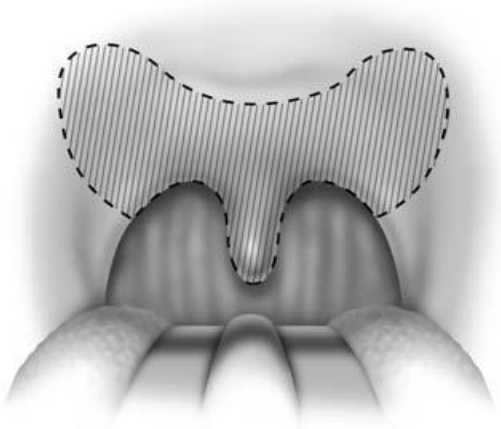


Figure 11:Incision site for Z plasty

Mucosa over the palate and uvula excised and palatal muscle exposed(fig 12)

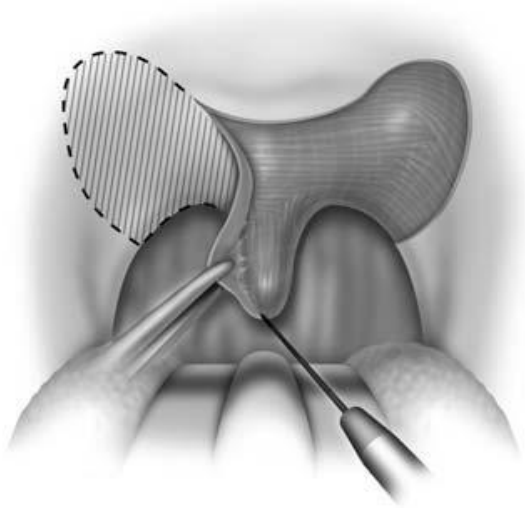


Figure 12: Palatal muscle exposed

Uvula is split in the midline(fig 13).

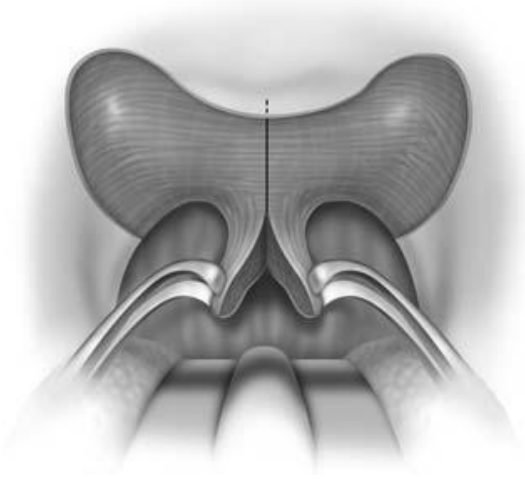


Figure 13: Splitting of uvula

Uvular flap along with palate is reflected laterally and closed in two layers. Ant and post pillar sutured(fig 14).

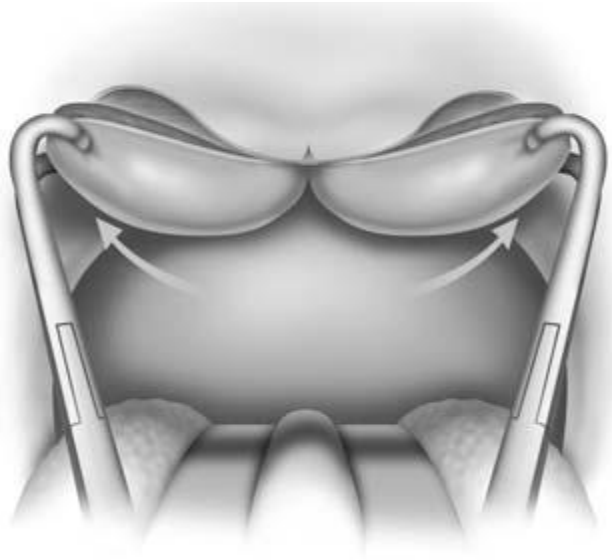


Figure 14:Wound closure

EXPANSION SPHINCTEROPLASTY

Indication: Lateral collapse at velum.

After tonsillectomy, palatopharyngeus is identified and its inferior end resected(fig 15).

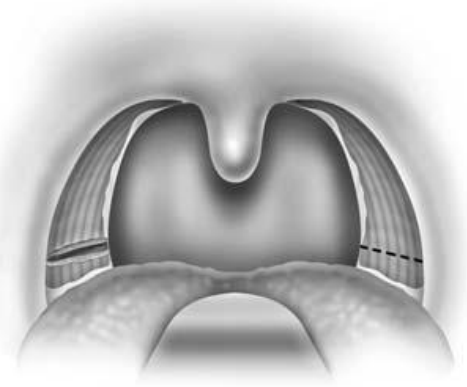


Figure 15: Incision in palatopharyngeus

Tunnel created in the softpalate and palatopharyngeus rotated posterolaterally and sutured. Uvuloplasty done. Anterior and posterior pillar sutured(fig 16).

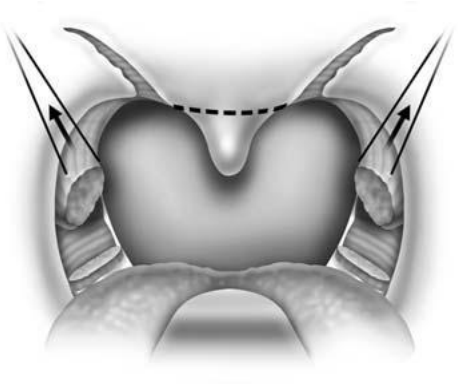


Figure 16: Rotation of palatopharyngeus

UPPP

Indication: Anteroposterior collapse at velum.

Incision site for UPPP(fig 17). Tonsillectomy done.

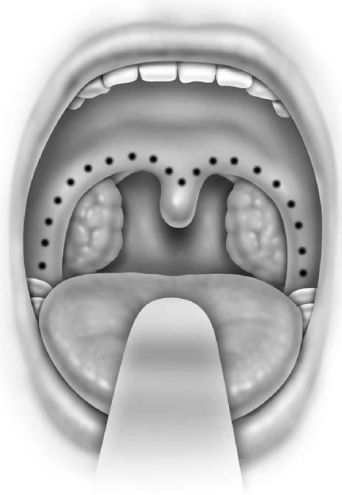


Figure 17: Incision

Incision made at the junction of post pillar and dorsal flap and pillar sutured to ventral flap.

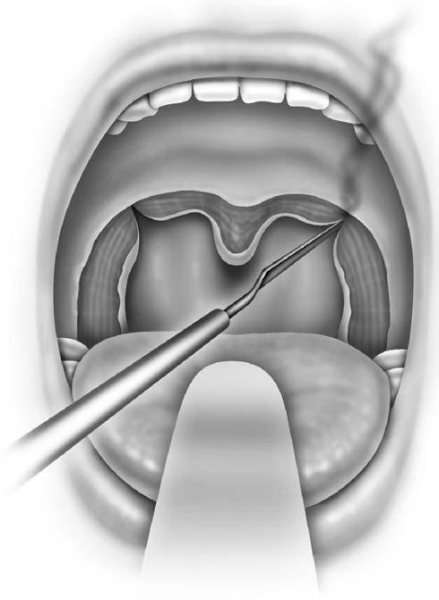


Figure 18: Incision at posterior pillar

In fairbank's technique of UPPP, final wound will be rectangular in shape (fig.19)

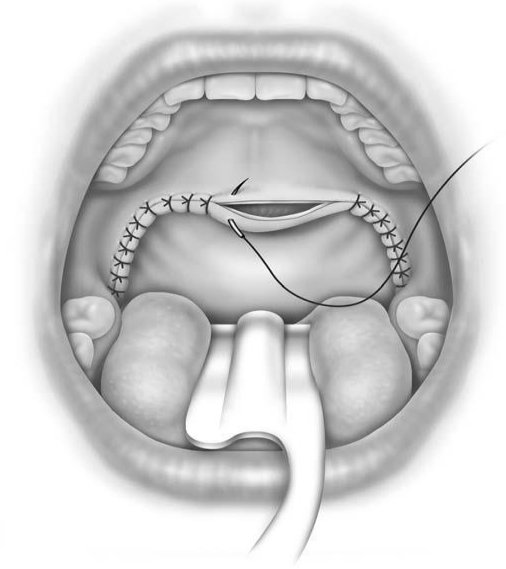


Figure 19: After wound closure

POST PROCEDURE

All patients were watched for complications like bleeding, pain(VAS score), VPI etc. Patients were given antibiotics, analgesics, mouth wash. Patients were given Ryle's tube feeding and discharged after 1 week. All patients were enquired about reduction in symptoms during subsequent follow up. At 3 months and 5 months polysomnography comprising of ECG, EEG, EOG, oxygen saturation, respiratory effort etc is done and AHI is calculated. All parameters were compared with pre surgical values and patients were categorized as responders or non responders. Non responders were thoroughly evaluated for the cause of failure and managed accordingly.

STATISTICAL ANALYSIS

Statistical analysis is done through SPSS 20 software. Descriptive statistical analysis done to summarize the baseline characteristics result. Mean, median and standard deviation has been calculated in descriptive statistics. Paired 't" test is used to compare the values of pre and post op BMI, oxygen saturation, ESS. Annova table is used to compare between baseline AHI and at 3^{rd}, 5^{th} month.

BASELINE CHARACTERISTICS

Of the 34 patients 29 (85.3 %) were males and 5(14.7%) patients were females. Mean age of the study population was 37.53±5.2. 8(23.5%) patients had a history of hypertension and 8(23.5%) had a history of diabetes. The level of obstruction after confirming with dynamic MRI and DISE were found to be in velum in 18 (52.9%) patients, velum and tongue in 9 (26.5%) patients, velum and nose in 3(8.8%) patients, oropharynx and elongated uvula in 2 (5.9%) patients, velum and uvula in 1(2.9%) and velum, nose and tongue together in 1(2.9%) patient.

Mean pre op BMI of these 34 patients were 29.02 ± 2.85. Mean Pre-op Epsworth scale was 16.41 ± 3.09. Mean Friedman score was 2.2 ± 0.76. Average pre op AHI was 41.73 ± 13.94. Average minimum oxygen saturation of 34 patients were 81.44 ± 4.85.

Table-1: Baseline characteristics of 34 obstructive sleep apnea (OSA) patients

Characteristic	Mean ±SD or Frequency, n(%)
Age	37.53±5.2
Male gender	29 (85.3 %)
Diabetes	8 (23.5%)
Hypertension	8(23.5%)
Level of obstruction	
Velum	18 (52.9%)
Velum and tongue	9 (26.5%)
Velum and nose	3 (8.8%)
Velum and elongated uvula	1 (2.9%)
Oropharynx and elongated uvula	2 (5.9%)
Velum,nose and tongue	1 (2.9%)
Pre-op BMI	29.02 ± 2.85
Pre-op Epsworth scale	16.41 ± 3.09
Friedman score	2.2 ± 0.76
Pre-op AHI	41.73 ± 13.94
Pre-op oxygen saturation	81.44 ± 4.85

Table-2: Age Group Distribution Of Study Participants

Age group	Frequency	Percentage
< 30 years	3	8.82
31 – 40 years	20	58.82
> 40 years	11	32.36
Total	34	100.0
Mean age	37.53 ±5.2	

Table-3: Sex Distribution of Study Participants

Sex	Frequency	Percentage
Male	29	85.3
Female	6	11.7
Total	34	100.0

Fig-20: Pie Chart Showing Sex Distribution of Patients

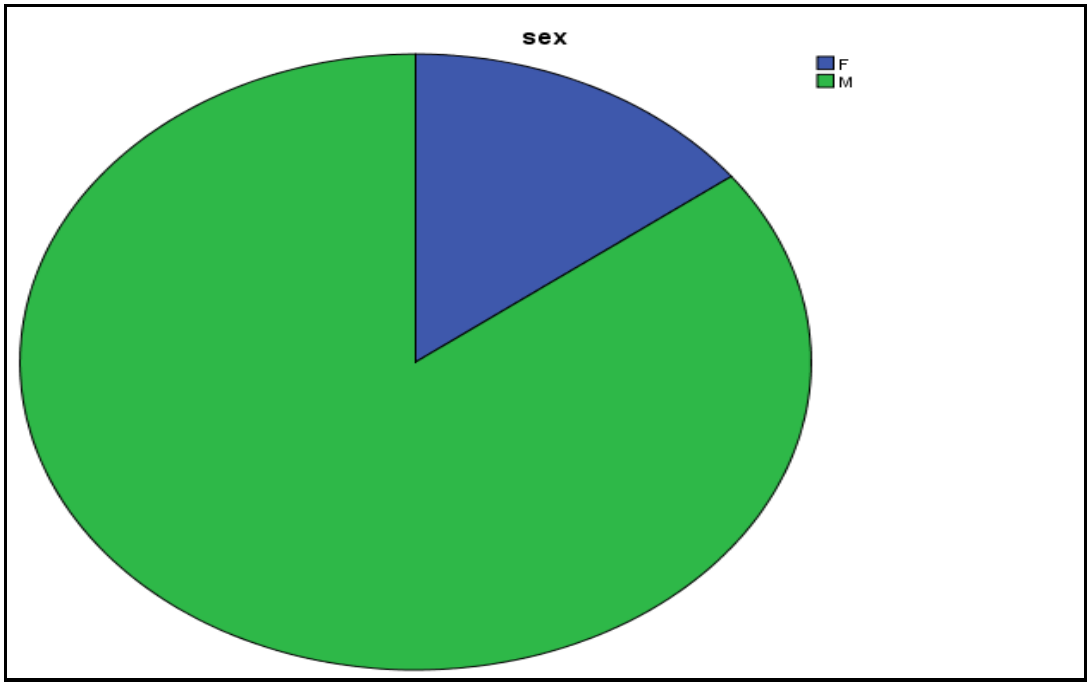


Table-4: Distribution of Study Participants By Bmi

BMI	Frequency	Percentage
18.5 – 24.99 [Normal]	3	8.82
25 – 29.99 [Overweight]	20	58.82
30 – 34.99 [Obese I]	9	26.47
> 35 [Obese II]	2	5.89
Total	34	100.0
Pre-op Mean BMI	29.02 + 2.85	

Table-5: Frequency Distribution of Study Participants by Comorbidity

Co-morbidity	Yes		No		Total
	Frequency	%	Frequency	%	
Diabetes	8	23.5	26	76.5	34 (100%)
Hypertension	8	23.5	26	76.5	34 (100%)

Fig 21. Bar diagram showing distribution of level of obstruction

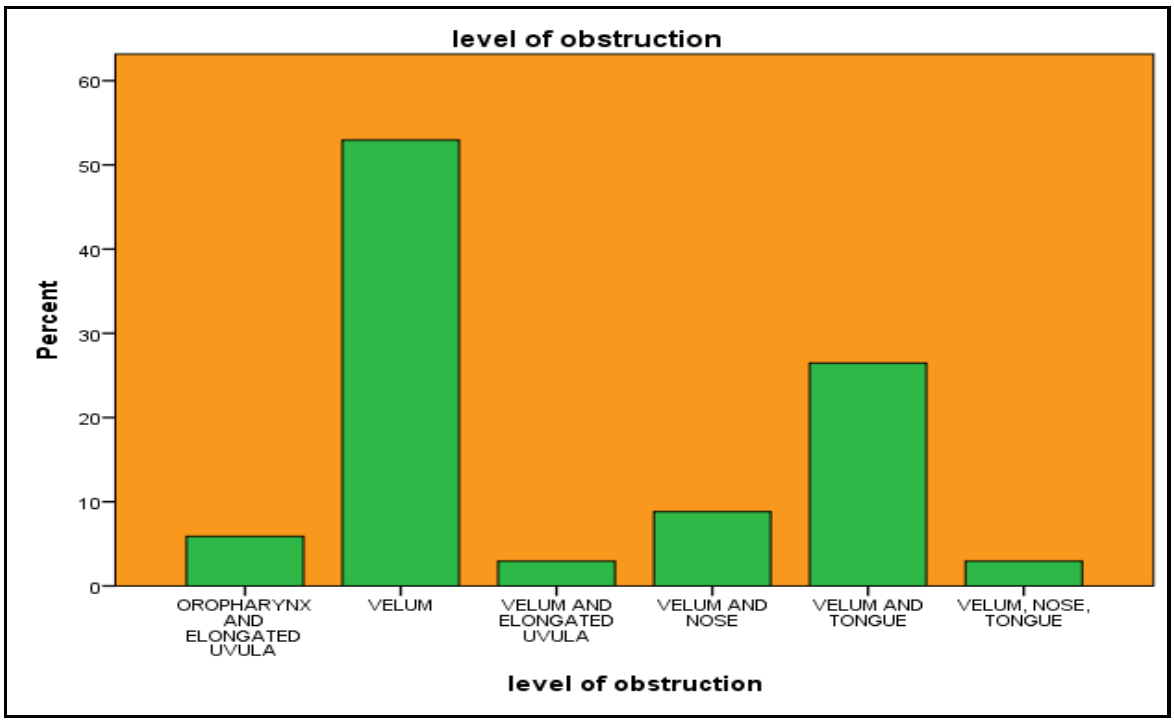


Table 6: Distribution of Study Participants by Direction of Obstruction

Direction of obstruction	Frequency	Percentage
Circumferential	14	41.2
Circumferential (Velum) & AP (Tongue)	8	23.5
Lateral wall	10	29.4
Lateral wall (Velum) & AP (Tongue)	2	5.9
Total	34	100.0

Table-7: Frequencies of different type of surgeries done in 34 OSA patients

Type of surgery	Frequency, n(%)
Zetaplasty	7 (20.6%)
Expansion sphincteroplasty	7 (20.6%)
UPPP	5 (14.7%)
Multilevel surgery	13(38.2%)
Tonsillectomy and uvuloplasty	2 (5.9%)

Among 34 OSA patients 7(20.6%) patients underwent zetaplasty and ESP each, 5(14.7%) patients underwent UPPP , 2(5.9%) patients underwent tonsillectomy with uvuloplasty and 13 patients (38.2%) had surgery done at multiple levels.

Fig-22: Bar diagram showing various types of surgery in 34 osa patients

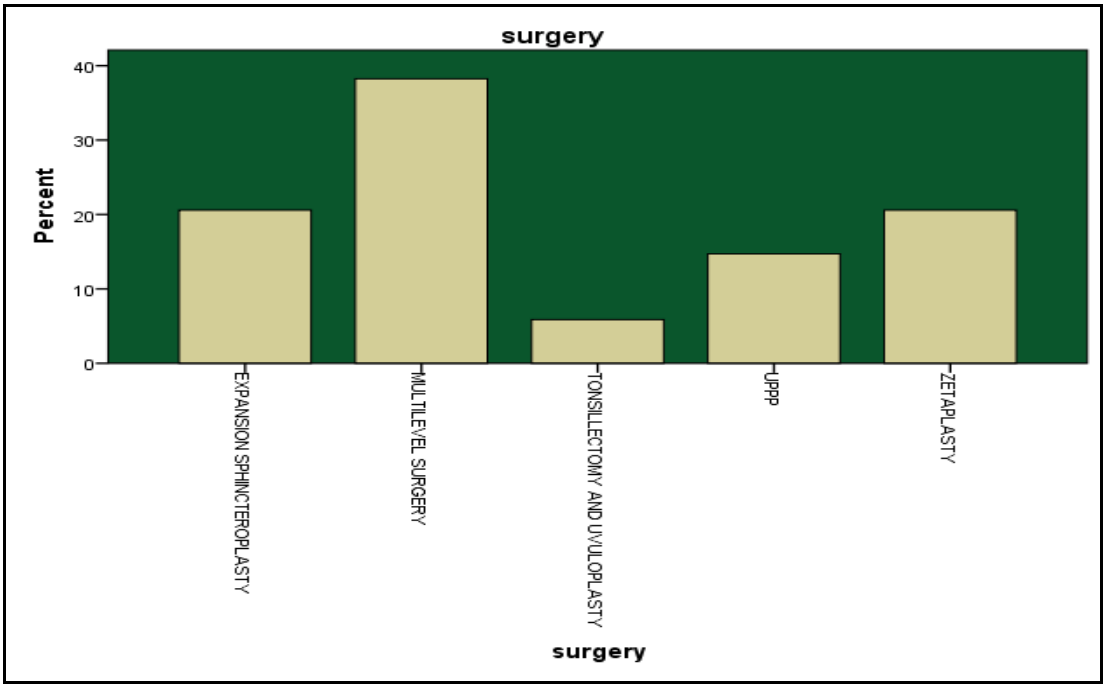


Table-8:Parameters Before and After Surgery In 34 Patients

Characteristics	Baseline	After 5 months of surgery	Mean difference	P value
BMI	29.02±2.85	27.52±2.96	1.49	<0.001
Epsworth score	16.41±3.09	5.14±3.41	11.26	<0.001
Oxygen saturation	81.44±4.85	93.02±4.18	11.58	<0.001

Mean BMI before and after surgery was 29.02±2.85 and 27.52±2.96 respectively with mean difference 1.49 which is statistically significant. Mean ESS of 34 OSA patients before and after surgery was 16.41±3.09 and 5.14±3.41 respectively with mean difference of 11.26 which is statistically significant. Average minimum oxygen saturation before and after surgery was 81.44±4.85 and 93.02±4.18 which is also statistically significant with mean difference of 11.58.

Fig-23: Box-whisker plot showing pre and post –op BMI

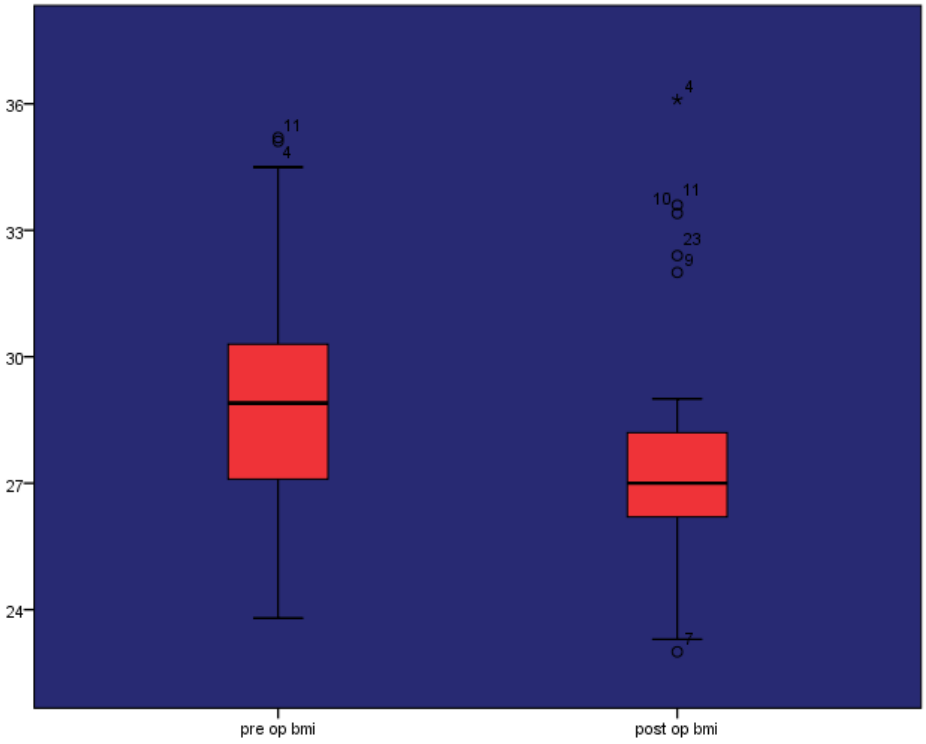


Fig-24: Box-whisker plot showing pre and post-op difference in epsworth sleepiness scale

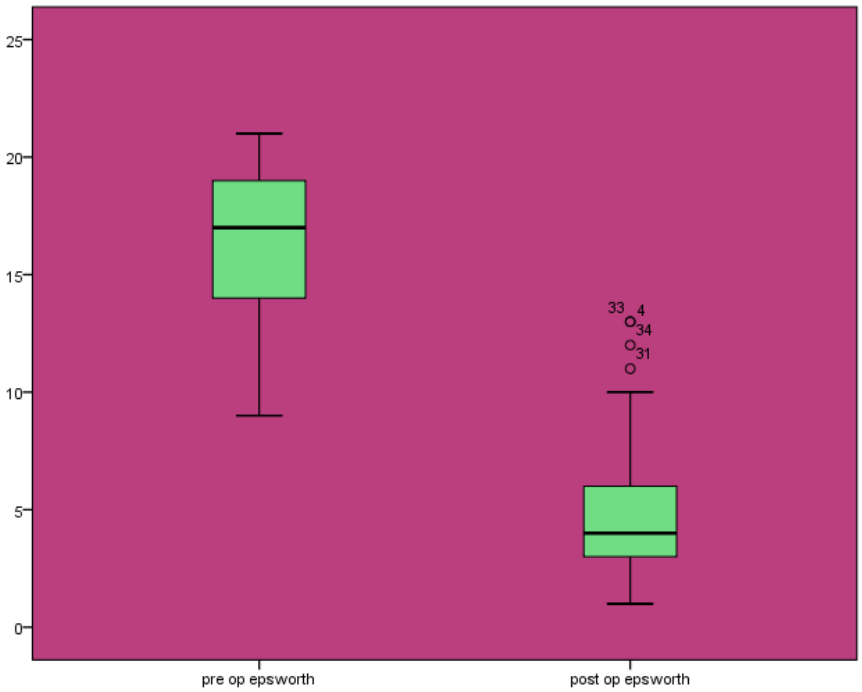


Fig-25: Box-whisker plot showing difference in oxygen saturation before and after surgery

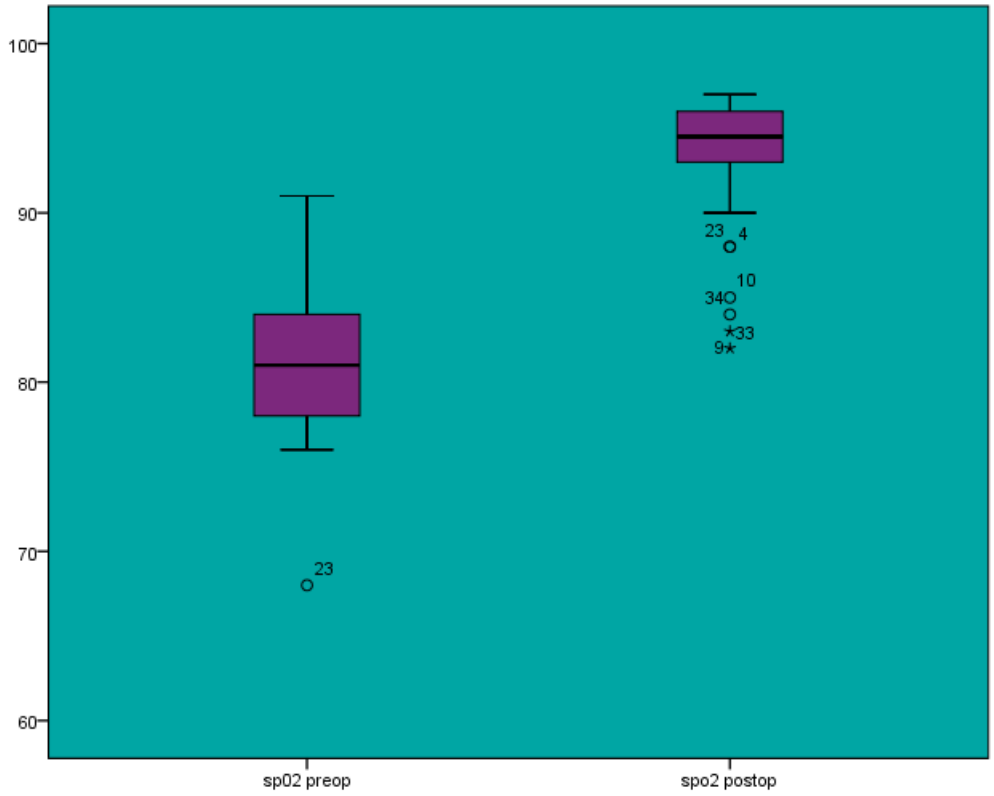


Table-9. Mean value of AHI before and after surgery at 3 and 5 months.

AHI at baseline	At 3rd month	At 5th month	P value
41.73±13.94	16.92±9.45	15.77±9.13	<0.001

Fig-26: Box-Whisker Plot Showing Improvement in AHI after Surgery

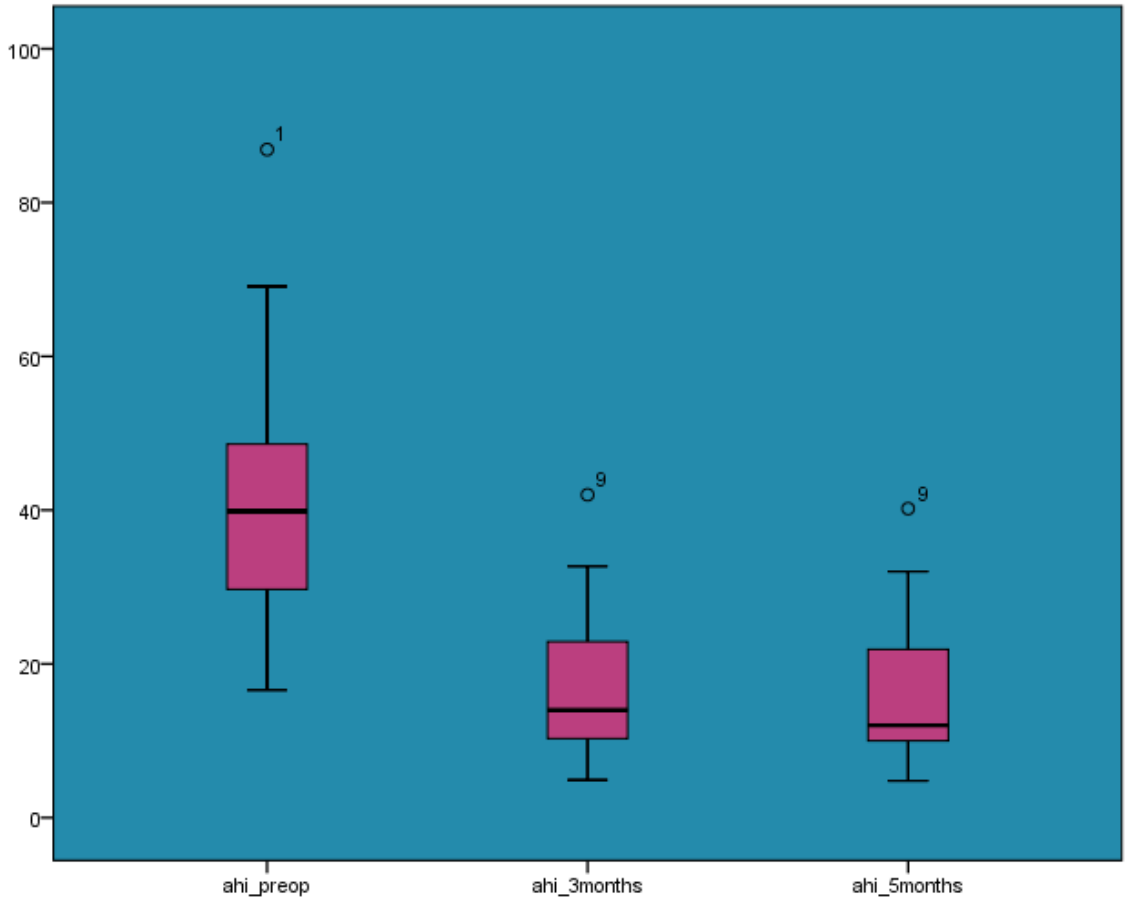


Fig-27: Profile plot showing estimated mean change in AHI over the time after surgery

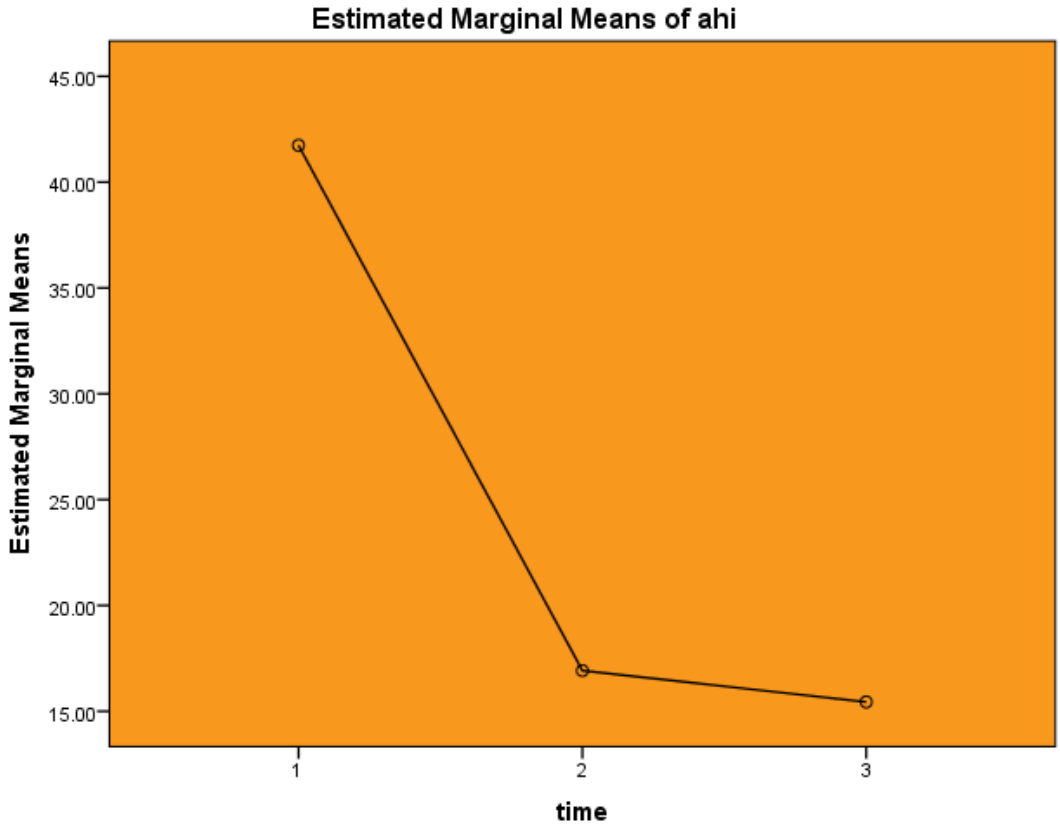


Table 10: Association Between Different Type of Surgery and Polysomnagraphic Findings

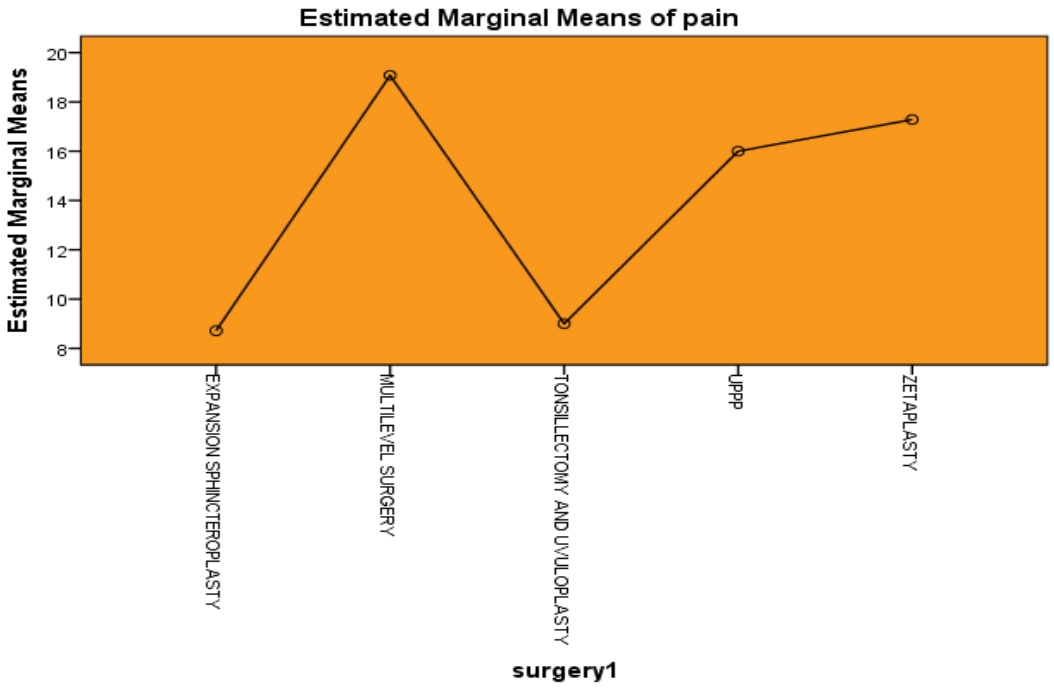
Surgery	EPSWORTH sleep scale [Mean + SD]		Oxygen saturation [Mean + SD]		AHI [Mean + SD]	
	Baseline	Follow up 5 months	Baseline	Follow up 5 months	Baseline	Follow up 5 months
Expansion Sphinteroplasty	13.14± 2.91	3.28± 1.38	84.43± 5.16	95.00± 1.16	38.35± 11.37	11.14± 4.63
Multilevel surgery	17.62± 1.76	6.31± 3.35	80.08± 2.72	91.69± 4.29	43.81± 9.39	18.39± 10.03
Tonsillectomy & uvuloplasty	12.50± 0.70	1.00± 0.00	84.50± 6.36	96.50± 0.71	26.25± 3.04	5.95± 1.20
UPPP	18.20± 1.64	7.40± 3.21	79.80± 7.26	93.80± 3.49	38.81± 9.46	15.68± 8.92
Zetaplasty	17.29± 3.45	5.57± 4.54	81.29± 5.16	92.00± 6.00	47.77± 23.45	18.43± 10.17
Total	16.41± 3.09	5.38± 3.53	81.44± 4.86	93.03± 4.19	41.74± 13.94	15.77± 9.13

Table-11: Complications Reported Among Study Participants

Complications		Frequency	%
VPI	Yes	6	17.6
	No	28	82.4
Bleeding	Yes	4	11.8
	No	30	88.2
Ventilator support	Nil	34	100
Pain duration (Days) [Mean + SD]		15.53 + 6.08	

The incidence of temporary VPI is about 17.6% and bleeding is about 11.8%. The mean duration of pain is about 15.33 $\pm$ 6.08

Fig 28. Profile plot showing estimated marginal means of pain duration in various type of surgery



The mean pain duration among various surgeries with maximum duration in multilevel surgery followed by zetaplasty

Table-12: AHI Percentage Reduction Of Study Participants

AHI reduction (%)	Frequency	Percentage
< 50	6	17.6
51 - 60	3	8.82
61 - 70	10	29.4
71 – 80	13	38.23
> 80	2	5.8
Total	34	100

AHI reduction (%)	Frequency	Percentage
< 50	6	17.6
> 50	28	82.4
Total	34	100

Table-13: Post Op Spo_2 % Reduction In Study Participants

Spo2 (%)	Frequency	Percentage
< 90	6	17.6
> 90	28	82.4
Total	34	100

Table-14: Post-Op Epsworth Sleep Scale Reduction In Study Participants

EPSWORTH Sleep Scale	Frequency	Percentage
< 10	7	20.58
> 10	27	79.42
Total	34	100

Fig-29: Pie Chart Showing Success Rate after Surgery

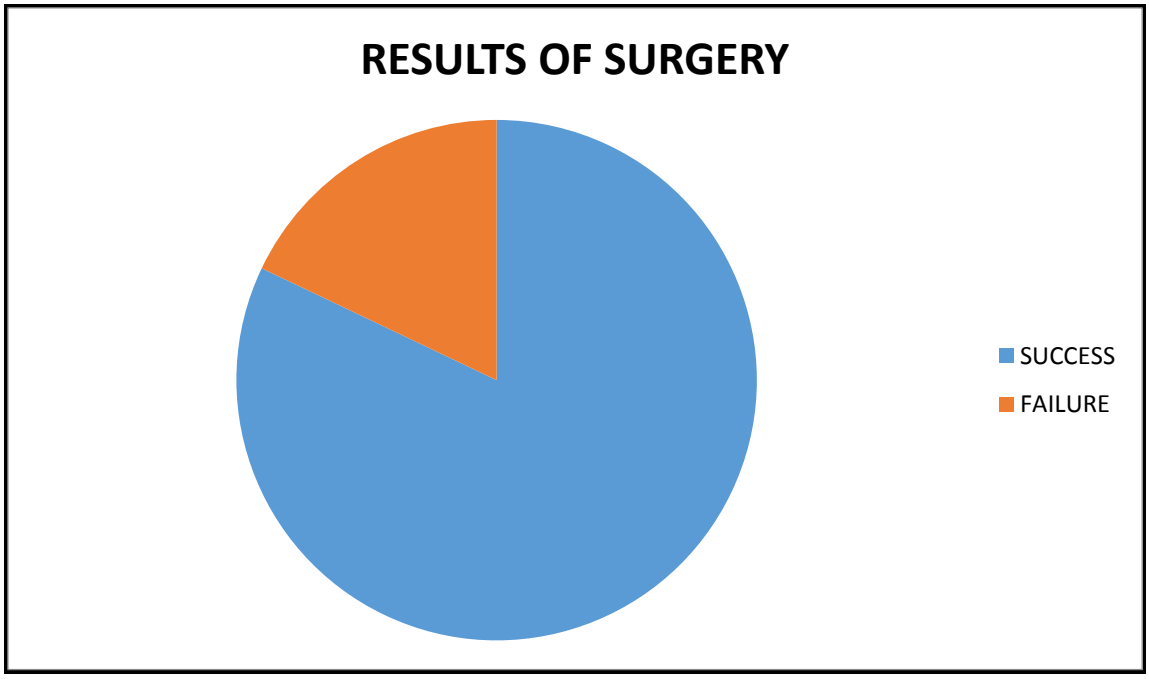


Table-15: Success and Failure after Surgery

Results	Percentage
Success	82.4
Failure	17.6

REVIEW OF LITERATURE

Obstructive sleep apnea is an entity of sleep disordered breathing . Patients with OSA presents with snoring, chocking spells, excessive daytime sleepiness and several other cardiovascular and neurocognitive sequelae. In order to avoid complications, patients should be initially advised for lifestyle modifications, CPAP and other oral appliances. Patients failing medical management are considered for surgery. Tracheostomy was the earlier surgery performed for OSA. Because of the morbidity associated with it, several surgeries addressing upper airway have evolved. Since the results of surgeries were inconsistent various studies had been conducted comparing the outcomes, complications and patient selection. The results of our studies were analysed and compared with other literature as follows

Akram khan ,MD et al[32] "conducted a study from jan 1988 to aug 2006 in 63 patients and analysed PSG and BMI in patients with OSA who are more than 18 years of age the mean age of the population was 42.1 comprising primarily men(81%) and mean pre op AHI is 62 who underwent UPPP . He concluded that AHI reduced to 50% the previous value in 32 patients with a success rate of 51%".

Mauro.B.M.Vieira et al[33] "conducted a study in 19 patients for treatment of snoring with or without apnea between nov 1998 to june 2000

who underwent palatal surgeries along with other surgeries. Patients were followed up for about 18 months. There were no case of nasopharyngeal stenosis and VPI and every patients improved after treatment. The study population had a main complication of odynophagia which lasted for about 10 days and there was one case of bleeding".

Thomas verse et al[34] "conducted a study of tonsillectomy as a treatment of OSA in adults. The study was conducted in 11 patients between aug 1996 to aug 1999.. All were subjected to PSG. Of which 9 patients had OSA of varying degrees. After tonsillectomy he found that 100% improvement in OSA of mild degree and 80.8% improvement in severe OSA without any complications. He concluded that in carefully selected patients tonsillectomy can be a safe option for OSA disorder".

Lin et al "conducted a systemic review/ meta analysis on outcome of patients with OSA. He pooled 58 studies and the OSA patients were treated with multilevel surgery for upper airway. Patients were followed up for a period of mean 7.3 months. They found that success rate was 66.4% and complication rate was 14.6%".

Staurt et al "conducted a study in 48 patients. He compared surgical outcomes of efficacy of modified UPPP with coblation channeling of tongue. Patients were followed up for 3 months. At the end of 3 months PSG was taken. Post op clinical assessment, sleep

questionnaire and BMI were compared. AHI reduced from 23.1 to 5.6 and ESS reduced to 5 from pre op value of 10.5".

Pang KP and Woodson et al "conducted a prospective RCT to assess the efficacy of ESP in OSA patients. They did a study in 45 adults with small tonsil, BMI < 30, friedman stage 2 and 3 with lateral wall collapse. They found that AHI reduced from 44.2 to 12 and lowest oxygen saturation improved from 78 to 85. He also found that in UPPP group AHI reduced from 38.1 to 19.6 and lowest oxygen saturation from 75.1 to 86.6. they found that success rate was 82.6% in ESP and 68.1% for UPPP. They concluded that ESP offers benefits in selected group of patients".

Vincente et al "conducted a prospective case series in 55 patients with severe OSA with multilevel upper airway obstruction who refused CPPP. Patient underwent UPPP and tongue base surgery. They found AHI improved in 78% of individuals and ESS decreased to < 11 in all patients after 3 year follow up".

Esclamado et al "conducted a retrospective study in 135 patients who underwent surgery for OSA, and found that complications occured in 13% of patients: 14 airway issues such as failed intubation and airway obstruction after extubation (1 of which resulted in death), 3 patients with hemorrhage, and 1 patient with arrhythmia".

Janson C et al[35] "conducted as study in 1997 in 25 patients and concluded that after UPPP Reduced prevalence of snoring and daytime sleepiness and reduction in AHI (mean [+/-SD], 40 +/- 26 to 21 +/- 21) at follow-up (P < .001). Sixteen patients (64%) were responders after 6 months and 12 (48%) at the long-term follow-up. Responders had a lower preoperative AHI (25 +/- 7) than non responders (48 +/- 29) (P < .05). None of the 7 patients with preoperative AHI of more than 40 were responders (P < .01). No difference was seen in preoperative body mass index, lung function, ventilatory response to carbon dioxide, computed tomography scan of upper airways, or change in body mass index between responders and nonresponders".

Keny P. Pang and Jin Keat Siow et al "conducted a study in 487 patients from jan 2007 to may 2010 on safety of multilevel surgery on obstructive sleep apnea. The mean AHI was 47.3. The study population mainly comprised of men. Multilevel surgery comprising nasal surgery, palate surgery(UPPP, ESP and anterior palatoplasty), tongue surgery was done. The results are, 7.1% of overall complication has occurred. Complications are postoperative desaturation, persistent hypertension, secondary hemorrhage, negative pressure pulmonary edema, upper airway obstruction requiring reintubation. They concluded that all OSA patients should be monitored in post anaesthesia care unit after surgery and based on the outcome in this period, patients should be shifted to general ward".

ANALYSIS AND DISCUSSION

This study was conducted in our institution in 34 OSA patients. After thorough evaluation with PSG, dynamic MRI, DISE patients were treated with various surgeries depending on the level of obstruction. Commonly UPPP, ESP, zetaplasty, tonsillectomy, tongue base reduction were done either alone or in combination.

Among 34 patients, 7 patients underwent zetaplasty and ESP, 5 patients underwent UPPP, 2 patients underwent tonsillectomy and uvuloplasty and 13 patients underwent multilevel surgery.

On statistical analysis on a study done in our institution, it was found that the study group comprised mainly male(85.3%). This data correlates with the study conducted by **Akram khan ,MD et al** where the population comprised of 81% men. This suggests that incidence of OSA is more common in male population.

The mean age was 37.15 ± 5.8 and the mean AHI was 41.88±15.35. The mean AHI in our study correlated with **Janson C et al and Pang**

KP where the mean AHI was 40.1 and 47.3 respectively suggesting that most of the OSA patients falls under severe OSA category.

The mean BMI in the study group comprises 29.02 coming under overweight category. This also correlates with **Pang KP and Woodson et al** where the study group had a BMI<30. This suggest that obesity plays a major causative factor in OSA patients.

The mean preop ESS is 16.41± 3.09 in our study group. This is similar to study conducted by **Kenny.P.Pang et al** in 487 patients where the mean ESS was 14.5. This suggest that significant proportion of population had symptoms of excessive daytime sleepiness.

Almost 100% of individuals had snoring preoperatively which correlates with the study conducted by **Whyte KF et al.** Snoring gets cured in 30 individuals and reduced in severity in 4 individuals after surgery as witnessed by partners.

The most common site of obstruction in our study group is velum(52.9%) followed by multilevel involving tongue and velum. This

study correlates with the study conducted by **Den Herder et al.** He reported that, of 127 patients, 63% had single level obstruction while only 37% had multilevel disease. But study conducted by **Riley et al** shows 93.3% (223 patients) were identified as having multilevel obstruction. Another study by **Abdullah van Hasselt et al** showed higher incidence of multilevel disease (87% of their 893 patient populations had multilevel obstruction).

On follow up PSG done at 5 months in our study, it was found that post operative AHI reduced to <50% than the preoperative value in 34 patients which is statistically significant and so the success rate was 82.4%. Whereas in a study conducted by **Akram khan et al and Elshaug et al** success rate was 51% and 51.5% respectively. This may be because of multilevel surgery done in our study and single airway surgery done in other studies.

In our study group AHI 8.82% of patients had 50-60% improvement in AHI . 29.4% patients had improvement in the AHI upto 60-70% of preoperative value and 38.23% of individuals had 70- 80% improvement in post op AHI.

In a study conducted by **Lin et al** success rate after multilevel surgery was 66.4% and in our study it is 82.4%. The increased success

rate in our study may be because of proper selection of patients by doing thorough pre operative evaluation using various available investigations.

On statistical analysis, two patients in our study underwent tonsillectomy with uvuloplasty as a sole surgical treatment for OSA. And these two patients improved well without any complications accounting a success rate of 100%. This result correlates with the study conducted by **Thomas verse et al and Stow et al,** where the result was 100% in mild group and 80.8% in severe OSA group. In our study the study population had a BMI of average 25, therefore patients with moderate OSA also responds to tonsillectomy if the obstruction is due to enlarged tonsil alone. But the disadvantage is lack of adequate population group.

Of the 34 patients, (17.6%) patients had VPI and 4(11.9%) patients had bleeding as a complication. This correlates with study conducted by **Esclamado et al**, but in a study conducted by **Mauro.B.M. et al** only one patient had bleeding.

In a study conducted by **Esclamado et al and Franklin et al** serious complications like death had occured(30 deaths in Franklin et al). In our study there was no case of mortality. Death in those above studies

were mainly due to postoperative airway edema. In our study because of proper airway management postoperatively mortality was avoided.

Among 34 patients, 28(82.4) patients had a postop oxygen saturation >90%, snoring reduced in 30 patients and ESS normalized(<10) in 27 patients. This suggests that after surgery there is both in subjective and objective improvement.

Incidence of hypertension and diabetes in our study population is about 23.5%(8 patients) each.

In those 6 patients who had failure, average BMI index was31.4 and there is no significant change between preop and post op BMI and thus suggesting weight reduction has a major role in the outcome of surgery.

The mean duration of pain in our study according to visual analogue scale is about 15.53 $\pm$ 6.08 which also correlates with study by **Mauro.B.M. et al.** The duration of pain varies with each surgery and it is more in surgery involving tongue base followed by zetaplasty

CONCLUSION

Obstructive sleep apnea which is an entity of sleep disordered breathing leads on to various systemic consequences, if left untreated. Thus in the study conducted in our institution patients of OSA who refused CPAP or failed after CPAP trail were vigorously investigated. After identifying the site of obstruction, patients were channelized to different surgeries addressing velum, tongue, tonsil and uvula. Significant proportion of patients improved both subjectively and objectively as determined by reduction in AHI from 41.73±13.94 to 15.77±9.13, reduction in ESS from 16.41± 3.09 to 5.14±3.41 and reduction in snoring. Since the complications of surgeries were anticipated preoperatively and managed accordingly, serious sequlae like airway compromise and deaths were avoided. Thus all surgeries were equally effective with proper preoperative investigations, appropriate surgery, anticipation and management of complications and continued lifestyle modifications.

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PATIENT CONSENT FORM

Title of the Project : EVALUATION OF PRE-OPERATIVE AND POST OPERATIVE POLYSOMNOGRAPHY FOR OBSTRUCTIVE SLEEP APNEA

Institution : Upgraded Institute of Otorhinolaryngology,
Madras Medical College,
Chennai – 600003.

Name : Date :

Age : IP No. :

Sex : Project Patient No. :

The details of the study have been provided to me in writing and explained to me in my own language.

I confirm that I have understood the above study and had the opportunity to ask questions.

I understood that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without the medical care that will normally be provided by the hospital being affected.

I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s).

I have been given an information sheet giving details of the study.

I fully consent to participate in the above study.

Name of the subject	Signature	Date
Name of the Investigator	Signature	Date

INFORMATION SHEET

- We are conducting a prospective cohort study on **"EVALUATION OF PRE-OPERATIVE AND POST OPERATIVE POLYSOMNOGRAPHY FOR OBSTRUCTIVE SLEEP APNEA"** at the Upgraded Institute of Otorhinolaryngology, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai – 600003.

- For snorers where the pathology is due to obstructive cause, surgery is one of the modality of treatment to make the patient free off symptoms and prevent the progression of disease and further complications.

- Polysomnography is a non invasive method of study which will help in identifying the severity of the disease and analyse the effectiveness of surgey using various parameters.

- At the time of announcing the results and suggestions, name and identity of the patients will be confidential.

- Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

- The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of Investigator **Signature of Participant**

Date :

ஆராய்ச்சி தகவல் தாள்

சென்னை ராஜீவ் காந்தி அரசு பொது மருத்துவமனையில் அதிக குறட்டை விடுதல் நோயின் காரணத்தை கண்டறியவும், நோயின் தன்மையை அறியவும் பாலிசோம்னோகிராபி (Polysomnography) எனும் பரிசோதனை செய்யப்படுகிறது. மேலும் நோயின் தன்மையைப் பொறுத்து அறுவை சிகிச்சை மேற்கொள்ளப்படுகிறது. அறுவை சிகிச்சைக்குப்பின் 3 மாதம் கழித்து மீண்டும் பாலிசோம்னோகிராபி எனும் பரிசோதனை மேற்கொள்ளப்படுகிறது.

இந்த பரிசோதனையில் பக்கவிளைவுகள் எதுவும் இல்லை.

நீங்களும் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம். அதனால் தங்களது நோயின் ஆய்வறிக்கையோ அல்லது சிகிச்சையோ பாதிப்புக்கு ஏற்படாது என்பதையும் தெரிவித்துக் கொள்கிறோம்.

முடிவுகளை அல்லது கருத்துக்களை வெளியிடும் போதோ அல்லது ஆய்வின் போதோ தங்களது பெயரையோ அல்லது அடையாளங்களையோ வெளியிடமாட்டோம் என்பதையும் தெரிவித்துக்கொள்கிறேம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின்பேரில்தான் இருக்கிறது. மேலும் நீங்கள் எந்த நேரமும் இந்த ஆராய்ச்சியிலிருந்து பின்வாங்கலாம் என்பதையும் தெரிவித்துக்கொள்கிறோம்.

இந்த சிறப்பு பரிசோதனைகளின் முடிவுகளை ஆராய்ச்சியின்போது அல்லது ஆராய்ச்சியின் முடிவின்போது தங்களுக்கு அறிவிப்போம் என்பதையும் தெரிவித்துக்கொள்கிறோம்.

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ஆராய்ச்சியாளர் கையொப்பம் பங்கேற்பாளர் கையொப்பம்

தேதி

PROFORMA

Name : **Date :**

Age : **Ip / Op no :**

Sex :

Occupation :

Presenting complaints :

History of presenting complaints :

S.no.	Complaints	yes	no	duration
1.	History of snoring			
2.	History of sleep awakening / insomnia			
3.	History of excessive day time sleepiness			
4.	History of impaired cognitive function			
5.	History of Hallucinations			
6.	History of increased weight gain			
7.	History of choking in sleep			
8.	History of nasal obstruction			
9.	History of mouth breathing			

Past history : Diabetes mellitus / Hypertension / Epilepsy / Asthma / Jaundice / previous history of accidents

Personal history : Diet , appetite , smoking , alcohol , chronic drug intake , bowel and bladder habits

Family history : married / unmarried

General examination :

Built :

Height :

Weight :

BMI :

Neck circumference :

Waist circumference :

Temperature ;

Pallor :

Cyanosis :

Jaundice :

Pedal edema :

Vitals ;

Pulse : BP : Respiratory rate :

Systemic examination :

Respiratory system :

Cardiovascular system :

Central nervous system :

ENT Examination :

Throat :

Oral cavity :

Gums :

Oral mucosa :

Floor of mouth :

Anterior 2/3rd tongue :

Hard and soft palate :

MALLAMPATI score :

Oropharynx :

Antr. Pillar :

Postr. Pillar :

Tonsil :

Posterior pharyngeal wall :

Indirect laryngoscopy :

Posterior 1/3rd tongue :

Vallecula :

Epiglottis :

Vocal cords and mobility :

Posterior pharyngeal wall :

Nose :

External contour :

Antr. Rhinoscopy :

Postr. Rhinoscopy :

Ear :

Pinna :

External auditory canal :

Tympanic membrane :

Neck :

Laryngeal contour :

Abnormal veins / scars :

Accessory muscles of respiration :

Investigations :

Blood investigations :

Thyroid functions test :

Diagnostic nasal endoscopy :

Video direct laryngoscopy :

x-ray Skull : Anteroposterior

Lateral

x-ray chest :

Patient name :

Diagnosis :

Management :

- Investigations
- Anesthesia
- Procedure

S.NO	NAME	AGE	SEX	BMI		EPSWORTH SLEEP		FRIEDMAN SCORE	COMORBIDITIES		AHI			MEAN SPO2		SNORING	
				PRE OP	POST OP	PRE OP	POST OP		DIABETES	HYPERTENSION	PRE OP	POST OP 3 months	POST OP 5 months	PRE OP	POST OP	PRE OP	POST OP
1	VIJAYAKUMAR	43	M	32.1	28	20	4	2	NO	NO	86.9	32	28	81%	95%	YES	NO
2	KUMARAKRISHNAPPA	47	M	28.7	25	19	3	1	NO	NO	69.1	22.2	20.4	91%	96%	YES	NO
3	JEYAVEERARAJAN	45	M	26.2	24.1	15	3	1	NO	NO	45.2	11.5	11	90%	96%	YES	NO
4	DINESH	31	M	35.2	36.1	21	13	2	NO	NO	48.4	30.2	30.1	83%	88%	YES	YES
5	SARASWATHY	35	F	26	23.3	12	2	1	NO	NO	58.9	22.9	20.1	83%	94%	YES	NO
6	MANIKANDAN	32	M	29.6	26.5	18	4	2	NO	NO	37.9	14.9	12.1	78%	93%	YES	NO
7	VIJAY	28	M	23.8	23	9	3	2	NO	NO	25.4	7.1	7.1	90%	96%	YES	NO
8	SOUNDARI	35	F	26.4	25.1	18	2	2	NO	NO	32.6	6.3	6.1	88%	95%	YES	NO
9	DEEPAK	36	M	32.1	32	18	10	3	YES	NO	58	42	40.2	78%	83%	YES	NO
10	UDAYAKUMAR	38	M	34.5	33.4	21	11	2	NO	YES	33	25	23	76%	85%	YES	YES
11	SURESHKUMAR	38	M	35.11	33.6	20	4	3	NO	NO	48.8	15.1	13.6	83%	94%	YES	NO
12	SRINIVASAN	43	M	30.3	28.2	13	6	2	YES	YES	38.3	12.3	11.8	84%	95%	YES	NO
13	SRINIVASAN	41	M	27.1	26.7	17	6	2	NO	YES	41.7	14.6	11.2	81%	97%	YES	NO
14	ADAISH	26	M	24.4	24	13	1	2	NO	NO	28.4	7.4	6.8	89%	97%	YES	NO
15	SANKAR	44	M	28.9	27.7	16	6	3	NO	YES	51.6	22.2	21.9	79%	90%	YES	NO
16	PACHIAPPAN	43	M	29.6	27	18	5	2	YES	NO	38.9	11.4	11.1	76%	94%	YES	NO
17	MANI	42	M	28.3	26.7	19	4	3	NO	YES	29.5	10.9	10.1	84%	93%	YES	NO
18	VENKATESAN	38	M	30.2	26.8	15	4	3	NO	NO	52.7	24.3	22.6	77%	93%	YES	NO
19	CHITHRA	32	F	27.4	27.2	11	3	2	NO	NO	28.2	9.9	9.4	78%	96%	YES	NO
20	KANDHAN	35	M	26.6	26.1	12	3	1	NO	NO	16.6	4.9	4.8	82%	97%	YES	NO
21	DILLIRAJAN	36	M	27.1	26.7	19	7	1	NO	NO	39.8	13.6	11.8	87%	96%	YES	NO
22	MASTHAN BASHA	32	M	24.2	24.1	13	1	2	NO	NO	42.6	9.1	8.7	84%	96%	YES	NO
23	ELLAPAN	41	M	32.3	32.4	20	10	2	YES	YES	48.6	32.1	31.5	68%	88%	YES	YES
24	MURUGANANTHAN	40	M	29.6	28.4	17	7	3	YES	NO	37.4	12.6	11.8	77%	95%	YES	NO
25	SHRINIVASAN	39	M	28.8	27.6	18	2	3	NO	NO	26.3	7.3	6.8	79%	94%	YES	NO
26	PRASANTH	26	M	26.4	26.2	12	1	2	NO	NO	24.1	5.1	5.1	80%	96%	YES	NO
27	SHANMUGAM	38	M	28.9	26.7	17	4	3	NO	NO	39.7	14.4	13.8	82%	92%	YES	NO
28	BANUMATHI	37	F	30.1	29	19	6	2	NO	NO	29.7	13.4	13.2	84%	96%	YES	NO
29	SUKUMAR	38	M	29.9	27.9	15	5	1	NO	YES	44.1	17.1	14.9	83%	95%	YES	NO
30	SHANKARI	41	F	31	27	14	4	3	YES	NO	39.9	16.8	15.2	78%	93%	YES	NO
31	CHINNATHA	34	F	27.8	27	16	11	2	NO	NO	28.6	10.3	10	79%	96%	YES	NO
32	RAVIKUMAR	40	M	28.2	27.9	17	3	3	YES	YES	47.4	12.6	11.9	81%	93%	YES	NO
33	GNANAVEL	39	M	29.1	26.2	18	13	4	NO	NO	48.3	32.7	32	77%	82%	YES	NO
34	GAJENDRAN	43	M	30.8	28.4	18	12	3	YES	NO	52.4	31.1	30.9	79%	84%	YES	YES

LEVEL OF OBSTRUCTION	DIRECTION OF OBSTRUCTION	SURGERY	SINGLE/ MULTILEVEL SURGERY	COMPLICATION			
				PAIN	VPI	BLEEDING	VENTILATOR SUPPORT
VELUM	CIRCUMFERENCIAL	ZETAPLASTY	ZETAPLASTY	20 days	NO	NO	NO
VELUM	CIRCUMFERENCIAL	ZETAPLASTY	ZETAPLASTY	21 days	NO	NO	NO
VELUM	LATERAL WALL	EXPANSION SPHINCTEROPLASTY	EXPANSION SPHINCTEROPLASTY	4 days	NO	NO	NO
VELUM AND NOSE	CIRCUMFERENCIAL	ZETAPLASTY AND INFERIOR TURBINATE ABLATION	MULTILEVEL SURGERY	18 days	NO	NO	NO
VELUM	LATERAL WALL	EXPANSION SPHINCTEROPLASTY	EXPANSION SPHINCTEROPLASTY	5 days	NO	NO	NO
VELUM	CIRCUMFERENCIAL	ZETAPLASTY	ZETAPLASTY	14 days	YES(TEMPORARY)	NO	NO
VELUM	LATERAL WALL	EXPANSION SPHINCTEROPLASTY	EXPANSION SPHINCTEROPLASTY	14 days	NO	NO	NO
VELUM	LATERAL WALL	EXPANSION SPHINCTEROPLASTY	EXPANSION SPHINCTEROPLASTY	7 days	NO	NO	NO
VELUM, NOSE, TONGUE	CIRCUMFERENCIAL(VELUM) AND AP(TONGUE)	ZETAPLASTY WITH TONGUE BASE REDUCTION WITH SEPTOPLASTY	MULTILEVEL SURGERY	24 days	NO	NO	NO
VELUM	CIRCUMFERENCIAL	ZETAPLASTY	ZETAPLASTY	24 days	NO	NO	NO
VELUM AND TONGUE	CIRCUMFERENCIAL(VELUM) AND AP(TONGUE)	ZETAPLASTY AND TONGUE BASE REDUCTION	MULTILEVEL SURGERY	21 days	YES(TEMPORARY)	NO	NO
VELUM	LATERAL WALL	EXPANSION SPHINCTEROPLASTY	EXPANSION SPHINCTEROPLASTY	7 days	NO	NO	NO
VELUM AND NOSE	LATERAL WALL	EXPANSION SPHINCTEROPLASTY AND INFERIOR TURBINATE ABLATION	MULTILEVEL SURGERY	10 days	NO	NO	NO
OROPHARYNX AND ELONGATED UVULA	LATERAL WALL	TONSILLECTOMY AND UVULOPLASTY	TONSILLECTOMY AND UVULOPLASTY	4 days	NO	NO	NO
VELUM AND TONGUE	CIRCUMFERENCIAL(VELUM) AND AP(TONGUE)	ZETAPLASTY AND TONGUE BASE REDUCTION	MULTILEVEL SURGERY	21 days	NO	NO	NO
VELUM AND SEPTUM	CIRCUMFERENTIAL	UPPP AND SEPTOPLASTY	MULTILEVEL SURGERY	14 days	NO	NO	NO
VELUM AND TONGUE	LATERAL(VELUM) AND AP(TONGUE)	EXPANSION SPHINCTEROPLASTY WITH TONGUE BASE REDUCTION	MULTILEVEL SURGERY	21 days	NO	YES	NO
VELUM AND TONGUE	CIRCUMFERENCIAL(VELUM) AND AP(TONGUE)	UPPP AND TONGUE BASE REDUCTION	MULTILEVEL SURGERY	21 days	NO	NO	NO
VELUM	LATERAL WALL	EXPANSION SPHINCTEROPLASTY	EXPANSION SPHINCTEROPLASTY	14 days	NO	NO	NO
VELUM	CIRCUMFERENCIAL	ZETAPLASTY	ZETOPLASTY	14 days	YES(TEMPORARY)	YES	NO
VELUM	CIRCUMFERENCIAL	UPPP	UPPP	24 days	NO	NO	NO
VELUM	CIRCUMFERENCIAL	ZETAPLASTY	ZETAPLASTY	14 days	NO	NO	NO
VELUM	CIRCUMFERENCIAL	UPPP	UPPP	14 days	NO	NO	NO
VELUM AND TONGUE	CIRCUMFERENCIAL(VELUM) AND AP(TONGUE)	ZETAPLASTY AND TONGUE BASE REDUCTION	MULTILEVEL SURGERY	21 days	YES(TEMPORARY)	NO	NO
VELUM AND TONGUE	LATERAL(VELUM) AND AP(TONGUE)	EXPANSION SPHINCTEROPLASTY WITH TONGUE BASE REDUCTION	MULTILEVEL SURGERY	21 days	NO	NO	NO
OROPHARYNX AND ELONGATED UVULA	LATERAL WALL	TONSILLECTOMY AND UVULOPLASTY	TONSILLECTOMY AND UVULOPLASTY	14 days	NO	NO	NO
VELUM AND TONGUE	CIRCUMFERENCIAL(VELUM) AND AP(TONGUE)	UPPP AND TONGUE BASE REDUCTION	MULTILEVEL SURGERY	21 days	YES(TEMPORARY)	NO	NO
VELUM	CIRCUMFERENCIAL	UPPP	UPPP	14 days	NO	NO	NO
VELUM AND TONGUE	CIRCUMFERENCIAL(VELUM) AND AP(TONGUE)	ZETAPLASTY AND TONGUE BASE REDUCTION	MULTILEVEL SURGERY	14 days	NO	YES	NO
VELUM	LATERAL WALL	EXPANSION SPHINCTEROPLASTY	EXPANSION SPHINCTEROPLASTY	10 days	NO	NO	NO
VELUM	CIRCUMFERENCIAL	UPPP	UPPP	7 days	YES(TEMPORARY)	NO	NO
VELUM	CIRCUMFERENCIAL	UPPP	UPPP	21 days	NO	NO	NO
VELUM	CIRCUMFERENCIAL	ZETAPLASTY	ZETAPLASTY	14 days	NO	NO	NO
VELUM AND TONGUE	CIRCUMFERENCIAL(VELUM) AND AP(TONGUE)	UPPP AND TONGUE BASE REDUCTION	MULTILEVEL SURGERY	21 days	NO	YES	NO

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To
Dr.M.Sivaranjani
Post Graduate in M.S.(ENT)
Madras Medical College/RGGGH
Chennai 600 003

Dear Dr.M.Sivaranjani,

The Institutional Ethics Committee has considered your request and approved your study titled **"EVALUATION OF PRE-OPERATIVE AND POST-OPERATIVE POLYSOMNOGRAPHY FOR OBSTRUCTIVE SLEEP APNEA" - NO.34122015.**

The following members of Ethics Committee were present in the meeting hold on **01.12.2015** conducted at Madras Medical College, Chennai 3

1. Dr.C.Rajendran, MD.,	:Chairperson
2. Dr.R.Vimala,MD.,Dean,MMC,Ch-3	:Deputy Chairperson
3. Prof.B.Sudha Seshayyan,MD.Vice Principal,MMC,Ch-3	: Member Secretary
4. Prof.B.Vasanthi,MD.,Inst.of Pharmacology,MMC,Ch-3	: Member
5. Prof.Raghumani,MS, Dept.of Surgery,RGGGH,Ch-3	: Member
6. Prof.Md.Ali,MD.,DM.,HOD-MGE, MMC,Ch-3	: Member
7. Prof.Baby Vasumathi,Director IOG, Ch-8	: Member
8. Prof.Ramadevi, Director, Inst. of Bio-Chem,MMC,Ch-3	: Member
9. Prof.Saraswathy, MD.,Director, Inst.of Path,MMC,Ch-3	: Member
10.Prof.Srinivasagalu,MD.Director,Inst.of Int.Med.MMC,Ch-3	:Member
11.Tmt.Rajalakshmi,JAO,MMC,Ch-3	: Lay Person
12.Thiru S.Govindasamy, BA.,BL,High Court,Chennai	: Lawyer
13.Tmt.Arnold Saulina,MA.,MSW.,	:Social Scientist

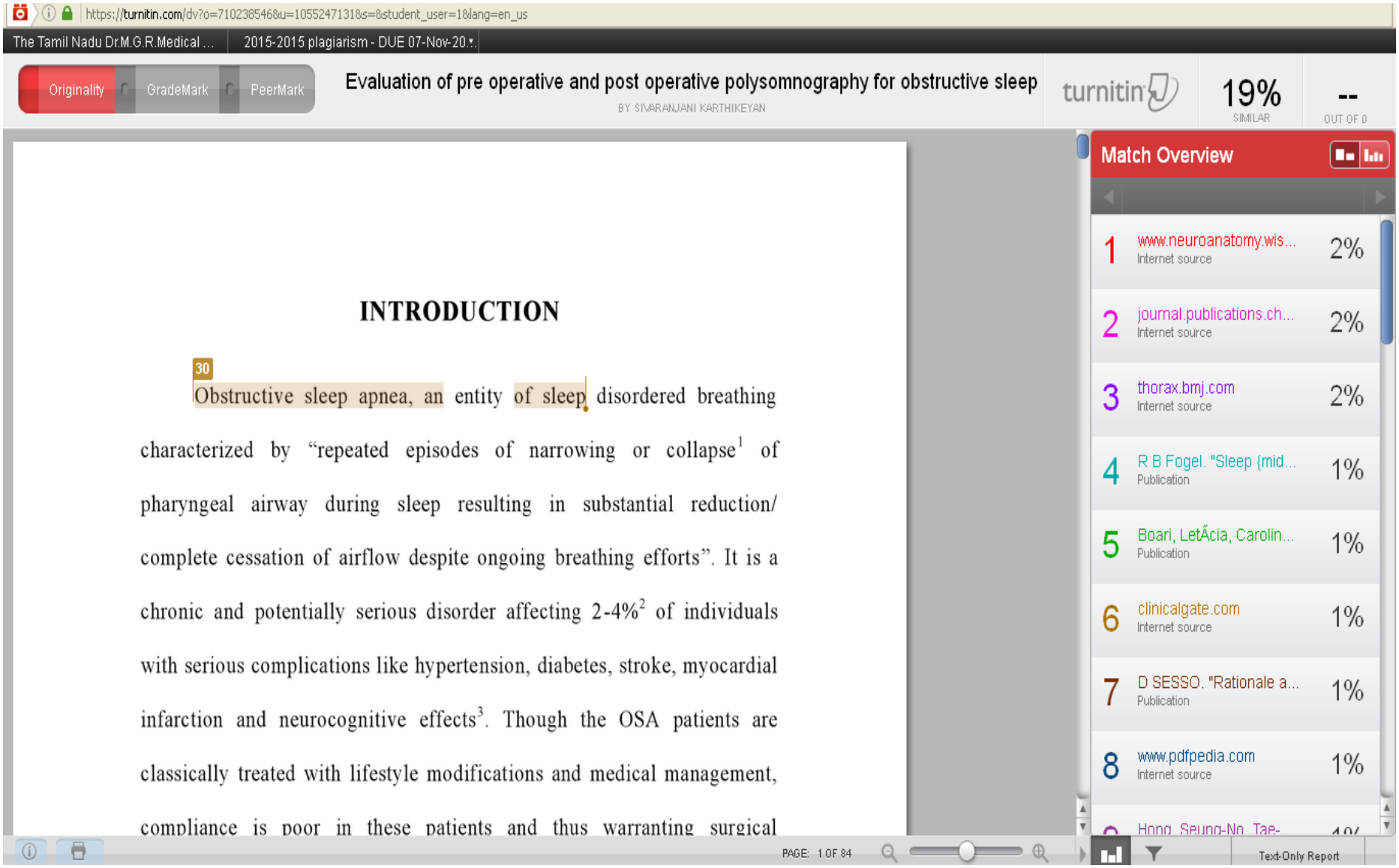
We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

24 JAN 2016

Member Secretary - Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003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INTRODUCTION
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Obstructive sleep apnea, an entity of sleep disordered breathing characterized by "repeated episodes of narrowing or collapse[1] of pharyngeal airway during sleep resulting in substantial reduction/ complete cessation of airflow despite ongoing breathing efforts". It is a chronic and potentially serious disorder affecting 2-4%[2] of individuals with serious complications like hypertension, diabetes, stroke, myocardial infarction and neurocognitive effects[3]. Though the OSA patients are classically treated with lifestyle modifications and medical management, compliance is poor in these patients and thus warranting surgical
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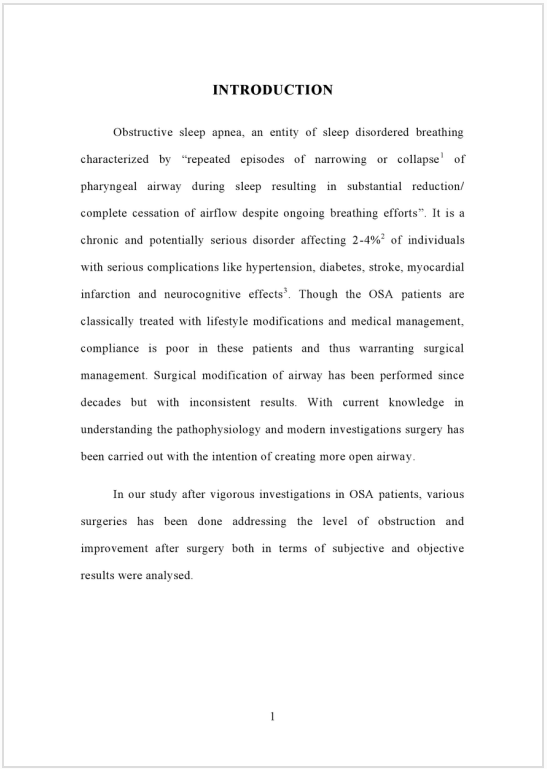

INTRODUCTION

Obstructive sleep apnea, an entity of sleep disordered breathing characterized by "repeated episodes of narrowing or collapse[1] of pharyngeal airway during sleep resulting in substantial reduction/ complete cessation of airflow despite ongoing breathing efforts". It is a chronic and potentially serious disorder affecting 2-4%[2] of individuals with serious complications like hypertension, diabetes, stroke, myocardial infarction and neurocognitive effects[3]. Though the OSA patients are classically treated with lifestyle modifications and medical management, compliance is poor in these patients and thus warranting surgical management. Surgical modification of airway has been performed since decades but with inconsistent results. With current knowledge in understanding the pathophysiology and modern investigations surgery has been carried out with the intention of creating more open airway.

In our study after vigorous investigations in OSA patients, various surgeries has been done addressing the level of obstruction and improvement after surgery both in terms of subjective and objective results were analysed.

1